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Evolution of Modern Surgery

TEXTBOOK OF SURGERY

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and 26 Color Plates*

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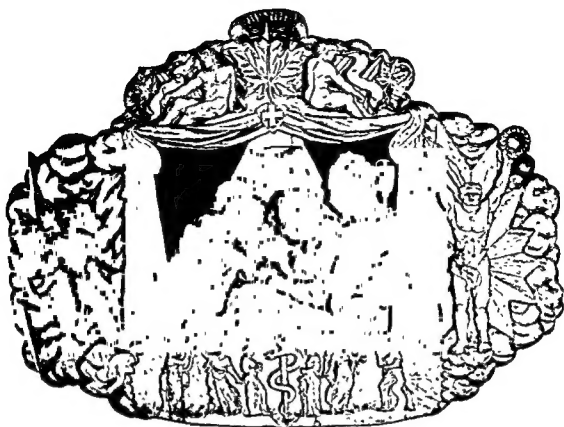
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This Textbook is dedicated to

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Surgeon-in-Chief, Royal Victoria Hospital

and

Chairman of the Surgical Department, McGill University

during whose tenure of office this

work was begun and with whose

assistance it was completed

FOREWORD

by

G. GAVIN MILLER, M.D.

There is no doubt that the great advances in surgery during the present century have created literature unprecedented in volume. It is frequently stated that surgery has advanced more in the last fifty than in the previous two thousand years. At the turn of the century, general surgery included many fields which today constitute the surgical specialties. Thus neurosurgery, thoracic surgery, cardiovascular surgery, and plastic surgery have developed, with great increase in detailed knowledge and refinement of technique.

Experimental research and clinical investigation have been diligently carried on in every medical center on the continent. From the voluminous literature which has developed, it has been difficult at times to select those ideas which should be put into practice, since a too hasty acceptance of some ideas could be harmful, whereas if too tardy, progress might be hindered. Careful appraisal of recent work is most important as changes are recorded so frequently. The head of a surgical department has an important responsibility in supporting with enthusiasm some of the new ideas suggested and, on the other hand, criticizing and even forbidding the practice of others. On what criteria can these judgments be made? New ideas, if they are to work in surgery, must be based on the sound principles of anatomy, physiology, biochemistry, pathology, and on broad clinical experience. When operative procedures are devised which are not based on such fundamentals, their practice is of short duration and they are soon abandoned.

The undergraduate must be taught the principles of surgery, the appreciation of clinical signs and symptoms, and methods of examination, so as to arrive at an accurate diagnosis. He should know when surgery should be recommended and have a fair idea of what is the best surgical treatment so that he can refer his patient to the surgeon best qualified to carry out the definitive treatment. It is possible that sufficient practical training should be added to enable him to carry out minor surgical procedures efficiently and well, but even this he can acquire more readily in his hospital training. Two years of hospital training is the minimum any graduate should have before he undertakes general practice. This approach leaves most of the actual training to become a surgical specialist to the postgraduate years, and the duration of such training has increased to five or more years from the time of graduation. This is a long time, perhaps too long after eight years of university study, to ask any man to work with little or no financial remuneration, but it is the men who have taken this long training and passed the examinations set by the Boards of Surgery and by the Royal College of Surgeons of Canada who have made the practice of surgery safe on this continent. Thus through the years of work and study such surgeons have raised the standards of surgery with great benefit to the public.

Too many young men are still accepting a somewhat haphazard surgical training. The training should be graded. It should include the basic sciences, and in



PREFACE

The writing of a textbook to contain the principles of surgery and the amount of factual information required by students at the time of their graduation and during their early period as interns was proposed by me in this center several years ago.

In order better to understand the approach to be followed, student forums were held. These indicated that the students, following the present trends in visual education, wished a profusely illustrated volume. They asked that sections on the Eye, Ear, Nose and Throat, and on Gynecology, apart from that required in the differential diagnosis in abdominal conditions, be omitted, since separate texts were used in these subjects. It was also decided that anatomy, physiology, and pathology should be integrated in the text and that no attempt be made to cover in detail these fields, since they are more adequately dealt with in their respective books.

To ensure the integration of the materials, the sections have been written by members of the Surgical Department of the Royal Victoria Hospital and of the Departments of Neurosurgery, Obstetrics and Gynecology, and Pathology, all associated with McGill University. Great overlap and cooperative discussion were required to achieve this end. Many difficulties presented from the rapid specialization even within special fields and the recent great changes due to chemotherapy, anticoagulants, and improved surgical technique. Balancing the proportions of various sections has been a great problem, and it is hoped that further critical evaluation will assist in this respect.

A book of this type represents the collective support of many individuals, and on behalf of the contributors I would like to acknowledge our debt to Dr. G. Gavin Miller, our Surgeon-in-Chief, who has placed the facilities of his Department at our disposal; to Drs. J. C. Armour, A. L. Wilkie and C. A. MacIntosh, Senior Consultants in General Surgery, to Dr. Newell Philpott, Director of the Montreal Maternity Hospital, to Dr. Wilder Penfield, Director of the Montreal Neurological Institute, to Dr. Emerson Smith, Urologist-in-Chief, to Dr. F. A. H. Wilkinson, Anesthetist-in-Chief, to Drs. Carleton Peirce and E. C. Brooks, Senior Radiologists of the Royal Victoria Hospital, and to Dr. Lyman Duff, Director of the Pathological Institute, all of whom have given valuable advice and criticism in the respective sections. We are also indebted to Dr. J. C. Meakins and Dr. E. H. Mason of the Department of Medicine for assistance on medical problems.

The photographic work is largely the product of Harold Coletta of the Pathological Institute. The references have been checked by Mrs. F. D. Peart of the Medical Library. The manuscript has been meticulously prepared by Mrs. Max Slapack.

Great credit is due to Miss Helen MacArthur of the Department of Medical Illustration of this Hospital, who, during the past three and one-half years, has worked assiduously to prepare the illustrations for this textbook and who has also assisted greatly in its organization and integration.

The editor wishes to thank all his colleagues who have given up their leisure time to write their individual sections and to contribute to the correlation of the materials. It is our hope that this textbook will assist students at all stages in their investigation of the fascinating study of surgery.

H. F. MOSELEY, D.M.

the final years the resident should have ample scope for carrying out the most complicated surgical techniques. Constant teaching should continue at the bedside, at ward rounds, pathological seminars, staff ward rounds, and surgical meetings. Perhaps a description of the Diploma Course in Surgery as planned at McGill University would help some students in planning their postgraduate surgical training.

After a year of rotating internship, applicants are accepted in the Course. The first year is spent as a junior assistant resident in surgery rotating through the various surgical services. The second year is spent in the surgical experimental laboratories of the University. During this year they take a refresher course in surgical anatomy, attend the various colloquia in physiology, carry out a research problem, and attend the staff ward rounds in the several teaching hospitals. Every effort is made to help the men financially during this year. The third year they return to the hospital as first assistant residents. The fourth year is a travel year where every effort is made to find a position for them under a man of recognized authority in some surgical field. The final year they return to the teaching hospitals of McGill University as residents. During these clinical years, they spend some time at each of the three teaching hospitals. This may be criticized as too long a training, but the men can break off and pass their Board examinations at the end of the fourth year if they so desire. Our next problem is to find means to help these men finance themselves during the long period of training.

The increasing complexity of medicine and the numerous specialties have made medical care very expensive. Hospital costs have risen sharply. Added to these, the numerous tests and consultations have made illness a financial drain which few people can afford without the aid of insurance. Further, the former close relationship between patient and physician is rapidly being lost. These factors are important in the growing demand for a national health scheme. This demand will continue to grow unless every effort is made by doctors to diagnose and treat patients as economically as possible by the avoidance of unnecessary tests and hospitalization and by spending a little more time in cementing the most cordial relationship between physician and patient.

These remarks are made to indicate the need of sound medical education and of textbooks which will not only include the sound that is new, but exclude the unsound both old and new. The great increase in medical knowledge has forced medical schools constantly to enlarge the curriculum, so that today the student has little time for leisure and contemplation. The multiplicity of examinations puts a premium on memory and does not necessarily develop the capacity for critical appraisal of the materials presented to him by his teachers.

It is hoped that this text, based on sound principles, will supply the surgical information the student requires in order to graduate. It emphasizes two aspects of teaching, namely, visual aid, and brevity of description. It represents the cooperative effort of the younger men of the surgical staff of the Royal Victoria Hospital. Its preparation has been of value to the department in many ways. It has brought the surgeons into closer understanding and friendship. It has taken them to the library to revise their knowledge. It has made them work together as a team. It has been good for us. I hope it will be of value to medical students in this and in other centers.

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TEXTBOOK OF SURGERY



Textbook of Surgery

CHAPTER I

THE EVOLUTION OF MODERN SURGERY

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Modern surgery is the result of a gradual evolution of knowledge. As the other sciences developed they made contributions that were integrated and elaborated into the science of surgery. Modern surgery dates from the nineteenth century, before that time, surgery was mostly that of trauma and manipulations. The great advances that have occurred can be appreciated after a brief historical consideration.

The origin of surgery dates back several thousand years. According to Prunières and Paul Broca, there is evidence that trephining of the skull was practiced in the Neolithic or Polished Stone Age for epilepsy, insanity, and persistent headaches.

SURGERY BEFORE CHRIST

Concerning early surgery, that of the Egyptians was recorded in the Edwin Smith Papyrus written about 1700 B.C., which was apparently a copy of another papyrus written about 3000 B.C. Fracture of the clavicle was treated by placing the patient flat on his back with a sandbag between the shoulder blades. A depressed fracture of the skull with brain injury was recognized, and the depressed fragment was removed. In India, the Rig-Veda and Atharva-Veda (1500 B.C.) contain information of medicosurgical importance and evidence of experimental surgery, but mysticism inhibited the progress of surgery. In the later Brahmanistic period the dissection of animals and human beings was forbidden for religious reasons, therefore operative surgery was taught by making incisions on inanimate objects.

Greek surgery was not developed until the time of Hippocrates (460-370 B.C.). He understood the necessity for close observation. He taught that the observation of phenomena should be considered first, then judgment, then general propositions, and finally practical knowledge and technique. He omitted, however, one general principle, the experimental method. The Greek surgeons observed that curvature of the spine was either due to trauma or was found with tubercles of the lungs. They showed an early attempt at asepsis by cleansing their hands and nails and irrigating wounds with boiled water.

THE CHRISTIAN ERA

The surgery of the Roman period was recorded by Celsus (25 B.C.-A.D. 50) and Galen (A.D. 130-200). Celsus recorded that hemorrhage was controlled by ligature. Amputations for gangrene were performed. The influence of Galen was felt until the fourteenth century. He was the first experimental physiologist, and he understood the mechanism of respiration. In his work he practiced vivisection on animals, sectioned the spinal cord at various levels, and described the symptoms, divided the recurrent laryngeal nerve, and noted the resulting loss of voice. Galen was an anatomist but his dissections were made on apes and pigs because human dissection was not permitted in his time. However, he considered that suppuration was essential for wound healing. Progress ended with the death of Galen about A.D. 200. During the following Arabian period, surgery remained dormant but the works of Hippocrates and Galen



With John Hunter (1728-1793) surgery nged from a technique to a division of lical science. Hunter was a pioneer in parative anatomy, physiology, and pa-logy, and he founded surgical and ex-perimental pathology; his greatest work was the surgical pathology of the vascular em. He founded the Hunterian Museum ich contained 13,000 pathological speci-ns. In the following generation a group great surgeons arose, John Abernethy, lip Syng Physick, the father of American rgery, and others

ANESTHESIA

The introduction and development of an-esthesia preceded antiseptis and aseptis. By 50 anesthesia was in general use. Al-ough *ether* had been known since the six-enth century it was first used in surgical actice on March 30, 1842, by Crawford illiamson Long (1815-1878) but did not come popular until 1846. On October 16, 1846, William Thomas Green Morton (1819-1868), a dentist, successfully demonstrated e use of ether on a patient of John Collins 'arren at the Massachusetts General Hos-ital. Nitrous oxide was isolated in 1776 by seph Priestley. Humphry Davy (1778-1829) noted the effect of the gas when in-aled, and suggested its use during surgical perations, but never attempted to employ

In 1824, Henry Hill Hickman (1800-1830) used nitrous oxide on animals. Horace vells (1815-1848), a dentist, administered itrous oxide while extracting teeth in 1844. One of Wells' patients died during nitrous ide administration, after which he gave up is researches and the practice of dentistry. n 1847 *chloroform* (discovered in 1831) as first used by Sir James Y. Simpson on bstetrical cases. It soon became the usual nesthetic in Great Britain. Chloroform has een utilized more than ether in Europe hile the reverse is true in America. The se of local anesthesia came after antiseptis and aseptis. In 1883 cocaine was used to

abolish pain in eye operations. Halsted was the pioneer in local cocaine infiltration an-esthesia, and in 1885 his work was well estab-lished. Spinal anesthesia was introduced by August Bier in 1898. With the discovery of novocaine by Einhorn and its clinical use by Braun in 1905, local anesthesia came into general use.

THE LISTERIAN ERA

With conquest of sepsis in 1864 began the era of modern surgery. In that year Pasteur (1822-1895) proved that fermentation de-pended upon living organisms. In 1867 Joseph Lister (1827-1912) published his pa-per on "The Antiseptic Principle in the Prac-tice of Surgery." He laid the foundation for *asepsis* and *antiseptis*. In 1871 he introduced the carbolic spray which was in use until 1887. The purpose of the spray was to kill germs in the air. In 1880 he introduced the absorbable catgut ligature. The science of bacteriology was advanced by Robert Koch (1843-1910) when in 1878 he showed that each disease was due to a specific organism. This placed surgery upon a firm scientific basis. The antiseptis of Lister was replaced by aseptic methods when Bergmann in 1886 introduced sterilization by boiling and Halsted in 1891 introduced the use of rub-ber gloves for operations. Asepsis made pos-sible the extraordinary development of ab-dominal surgery at the end of the nineteenth century.

ROENTGEN RAYS

Following anesthesia, antiseptis, and asep-sis came the discovery of *Roentgen rays* (x-rays) in 1895 by Wilhelm Konrad Rönt-gen (1845-1923). X-rays were first used for the diagnosis of fractures and to visualize foreign bodies. The examination of internal organs soon followed, and finally they were employed for treatment.

Thus by the end of the nineteenth century the three great pillars were placed and mod-ern surgery was established.

were translated and carried on for posterity. This period is represented by the writings of Rhazes (850-923); Albucasis (936-1013); and Avicenna (980-1036)

The revival of surgery began in Italy, by the eleventh century the School at Salerno was well known. This school in time became famous throughout the world. It was the first European school to make the teachings of the Arabs, and thus the methods of the ancients, available to the continent. In 1180 Roger of Salerno wrote the "*Practica Chirurgiae*". His book was based on the Pantegni of Haly Abbas (fl 994) which was translated into Latin by Constantine (1010-1087). Thyroidectomy was described including preoperative systemic treatment with calined sponge and seaweed

As a result of war there developed a group of notable surgeons. In Italy there arose William of Salicet, Theodoric and Lanfrank. William of Salicet (1201-1277) recognized arterial bleeding and discarded the cautery for the knife. Theodoric (fl 1250) insisted that wounds should heal without suppuration. Lanfrank (d 1315) also attempted to get wounds to heal by first intention. In France, Henri de Mondeville and Guy de Chauliac were outstanding. Henri de Mondeville (1260-1320) insisted on cleanliness and sharp needles to get wounds to heal by first intention. He laid down high ideals for a surgeon and said, "he should not undertake any dangerous operation unless he is sure that it is the only way to avoid a greater danger—he should neither praise himself nor blame others and he should not hate any of his colleagues." Guy de Chauliac (1298-1368) wrote a popular textbook of surgery, but unfortunately taught that suppuration was necessary part of wound healing. In England, John of Arderne (1307-1380) was first to revive surgery. He differentiated between ulcer and cancer of the rectum.

In the thirteenth century the universities did not recognize the surgeons, but under exceptional circumstances gave a license to

practice surgery. As a result in Paris the College of St. Come came into being about 1350 and its members were referred to as the "surgeons of the long robe." The barber surgeons were called the "surgeons of the short robe" and performed barbering and phlebotomies. The physicians often tricked the "surgeons of the long robe" by employing barbers to do operations which the physicians directed. In London there existed in the middle of the fourteenth century a Fraternity of Surgeons. In 1540 the United Company of Barbers and Surgeons was formed by the union of the Gild of Surgeons and Company of Barbers. The surgeons did the surgery while the barbers performed tonsorial duties and phlebotomies.

The first real advance from early and medieval surgery was made by Ambrose Paré (1510-1590). He had an inquiring open mind and wrote excellent original works. He stopped the use of the cautery for hemorrhage in amputations and popularized the ligature. He performed amputations not as a last measure but as operations of choice. About this time modern anatomy was founded by Andreas Vesalius (1514-1564). His "*Humani Corporis Fabrica*" published in 1543 laid the foundation for modern surgery. In England, Thomas Gale (1507-1587), William Clowes (1540-1604), John Halle (1529-1568) and others did much to raise the status of surgeons. Organized courses in anatomy and surgery were given. In order to gain admittance into the United Company of Barbers and Surgeons, a thorough examination was required. A higher license in Surgery was offered after a more searching examination. The "*De Motu Cordis*" of William Harvey (1578-1657) published in 1628 made physiology a working science. This had a profound effect on surgery. Richard Wiseman (1620-1676), who became the father of modern British surgery published "*Several Chirurgical Treatises*" in 1676, written for surgeons, thus showing an early attempt at postgraduate teaching.

paring large quantities of heparin. Between 1935 and 1942, Gordon Murray in Canada and Crafoord in Sweden simultaneously and independently showed that thrombosis can be prevented by heparin postoperatively. Dicumarol was described, isolated, and synthesized by Link in 1940 and came into clinical use in 1941. It is interesting that the discovery of this agent was due to Schofield, who in 1922, in the Canadian Veterinary Record, described a disease in cattle in which there was a tendency to bleed after eating spoiled sweet clover. The use of the anticoagulants after major surgical procedures for prophylaxis and treatment has diminished the incidence and mortality of pulmonary thrombosis and embolism. With these two agents greater safety in vascular and cardiac surgery became possible, and considerable progress has been made. Twenty-five years ago cardiac surgery was limited to fairly simple procedures. Today, operations on the valves of the heart, operations for improving the coronary circulation and the correction of congenital defects of the heart and great vessels have become frequent. A method of increasing the coagulability of the blood and the prevention of postoperative hemorrhages in obstructive jaundice became available with the advent of vitamin K ("Koagulations-Vitamin"). This was discovered by Dam in 1934 and was used clinically in 1938.

Technical Advances

As a consequence of World War II, in addition to the discovery and development of the sulfonamides and the antibiotics, improved methods of treatment have come into use. More fractures are now treated by internal fixation with inert metals, and bone banks are being established to extend the use of bone grafts. In plastic surgery better operations have been devised and the handling of skin grafts has become more precise. Refined methods of blood transfusion and the combating of shock have been developed. Banks of preserved blood and plasma

are now in general use. While no significant advances have been made in the etiology of cancer, cytological techniques for the early detection and radical operations for complete removal of the growth have been perfected. Radioactive isotopes are being utilized as a method of investigation of disease and in selected instances for treatment. One of the great developments of the twentieth century was the surgery of the brain, pioneered particularly by Harvey Cushing (1869-1939). The field of thoracic surgery was pioneered in the 'twenties by Archibald who advanced the methods advocated by Sauerbruch. He was one of the first surgeons on this continent to operate for pulmonary tuberculosis, and his work helped greatly in the development of the surgical treatment of this disease as well as thoracic surgery in general.

The Training of a Surgeon

While surgery has developed by a process of evolution from an art to a science, the training of the surgeon has also changed. The surgeon should have a thorough grounding in the Humanities before he seeks a knowledge of the basic sciences, physiology, anatomy, biochemistry, bacteriology, and before he attempts to coordinate pathology, roentgenology, and internal medicine. Sir Heneage Ogilvie stated, "There are at least four stages in the forming of a surgeon: he must be found, he must be qualified, he must be trained, and he must be given opportunities." Of the four the training stage requires further consideration.

The doctor who plans a career in surgery must have a definite schedule of postgraduate training. In addition the course should be planned to meet the requisites of the various certification boards. A suggested minimum course should include six months in pathology, one year of internship in an approved hospital, and two and one-half years in general surgery in an approved hospital. Those who plan to undergo a more thorough training and receive a Fellowship

SURGERY IN THE TWENTIETH CENTURY

The twentieth century may be divided into two periods, 1900 to 1936 and 1936 until the present. The first period was influenced by World War I and the discovery of *insulin*. The second period was influenced by World War II and the advent of the *sulfonamides*, the *antibiotics* and *transfusions*.

As a result of World War I antiseptics, germicidal but less destructive to tissues, were developed. Débridement of wounds and prophylactic inoculation against tetanus were introduced. In 1921 Banting and Best isolated *insulin*. This discovery had a profound influence on the surgical treatment of diabetics. Diabetic gangrene was more easily controlled and operations on the diabetic could be performed with the same degree of safety as on the nondiabetic.

The Age of Chemotherapy and the Antibiotics

The present era was ushered in with the use of the *sulfonamides* in the treatment of infections in 1936. Gelmo in 1908 synthesized para-amino benzene sulfonamide (*sulfanilamide*) which was used in the dye industry. It was not until 1933 that Foerster in Germany reported upon the first clinical use of a sulfonamide compound known as *Prontosil*. Domagk in 1935 reported the first experimental work on *Prontosil* in which he proved that it prevented fatal hemolytic streptococcal infections in mice. Simultaneously there appeared clinical confirmation of Domagk's work in that the drug was effective in septic hemolytic streptococcal angina, erysipelas, puerperal sepsis, and other infections. In 1936 Colebrook and Kenny in England reported that *Prontosil* and a variant *Prontosil S* was of definite value in the treatment of puerperal sepsis. Interest in these compounds in the United States did not appear until 1936 when investigations were begun by Mellon and his associates, Rosenthal and Bauer, and Long and Bliss.

Long and Bliss, formed the hypothesis that *sulfanilamide* acted directly upon the invading microorganism and that this action was primarily one of bacteriostasis. They were convinced that *Prontosil*, *Prontosil S* and *sulfanilamide* were of definite therapeutic value in streptococcal infections.

During the years of World War II there followed *sulfapyridine* in 1939, *sulfathiazole* in 1940, *sulfadiazine* in 1941, and *sulfamerazine* in 1943. Then followed *Sulfasuxidine* and *Sulfathalidine*. New sulfonamide compounds are continually being developed.

In 1929 Alexander Fleming discovered *penicillin* and suggested that it might be an effective bacteriostatic agent to penicillin-sensitive organisms. Fleming's discovery remained unnoticed until 1940 when interest in the clinical use of *penicillin* was aroused following the investigations of Chain, Florey, and their associates at Oxford University. Since that time, the other antibiotics, *streptomycin*, *aureomycin*, and *chloramphenicol* were discovered. From 1940 there has been greater progress in the control of infections than has taken place in all previous medical history. The use of the *sulfonamides* and the antibiotics has made practical many surgical procedures that before were not possible with any degree of success.

In intestinal cancer, the sterilization of the bowel before operation has made possible the one-stage instead of the multiple-stage resection. Hospitalization has been shortened and the mortality rate reduced to one-tenth the former rate. Total gastrectomy, total pancreatectomy and total colectomy are now often performed.

The Control of Blood Coagulation

The anticoagulants *heparin* and *Dicumarol* have been two extremely effective methods of therapy for venous thromboembolic disease. As late as 1934 surgery was helpless against thrombosis. Heparin was discovered by McLean in 1916. In 1933 Best and his co-workers solved the problem of

CHAPTER II

REACTION TO INJURY AND REPAIR

THEO. R. WAUGH, M.D

To continue in a state of health, a living organism must be in equilibrium with its environment. This is made possible through the irritability of the living cell. In the lower forms, such as an ameba, this would appear to be a relatively simple process, though we know very little of the finer details of such activity. In the higher forms, such as man, it becomes exceedingly complex because one has to deal not only with changes in the environment external to the body, but also, which is much more important, with alterations in the milieu of the internal cells of the body. In the final analysis it is the inability to maintain this latter equilibrium which constitutes disease. And if not restored, will eventually lead to death.

During the evolution of higher metazoic organisms, the adaptation to environmental changes has been profoundly affected in two ways, one in a sense deleterious and the other, for the most part, beneficial. With differentiation and specialization, many types of cells of the body have sacrificed much of their ability to respond favorably to local alterations and have become exceedingly vulnerable. On the other hand, this same specialization has made possible the development of protective structural changes in the organism. Examples of such are furnished by the integument, the sensory nervous system alert to external stimuli and a number of complex reactions, some of which are localized and others much more generalized. These tend to restore equilibrium and constitute reaction to injury.

CAUSES OF INJURY

The various causes of injury are the etiological factors of disease. With some of

these, such as *trauma*, the manner in which they alter the environment of cells and tissue beyond the point of physiological adaptation is perfectly obvious, but with others, as *bacterial infections*, it is more complicated though comprehensible. In the case of certain injurious factors, such as *carcinogenic agents*, the method of action is purely hypothetical and we have a considerable number of diseases termed *idiopathic* in which the etiological agent itself is quite unknown.

A fairly sharp division can be drawn between extrinsic and intrinsic causes of disease. The *extrinsic factors* may be further divided into (1) physical, such as mechanical trauma, heat, cold, radiation and changes in atmospheric pressure; (2) chemical, such as poisons, excesses or deficiencies of diet and some carcinogenic agents; (3) living organisms ranging from viruses and bacteria to large parasitic forms, and finally; (4) foreign inanimate substances which enter by injection or through the various orifices of the body.

The *intrinsic causes* of disease are by no means as clearly defined, and in them no doubt lie the hidden explanations of many idiopathic conditions. Here must be included (1) the normal ageing of the body which is bound to occur quite apart from extrinsic influences; (2) constitutional anomalies or deficiencies which may or may not have an hereditary basis; (3) psychosomatic factors; (4) changes of structure or function resulting from a previous disease which prepare the way for the development of another.

REACTION TO INJURY

The reactions of the body and tissues to an injurious agent must of necessity vary according to the nature of the injury and

in one of the Colleges, should in addition include a year in one of the basic sciences or experimental surgery, a year at various clinics, and a residency in surgery.

Finally improved methods of visual surgical education are being developed by means of the motion picture and television. The surgical societies and the medical schools are gradually building up libraries of valuable teaching films. In some centers operations are being televised. The advantages of this medium are obvious.

In addition to a scientific training, a surgeon must possess and develop fine qualities of mind and body. Hutchison has beautifully described the attributes of a surgeon in his address to the students of the London Hospital Medical College, entitled "Seven Gifts"

"Good health . . . that sort of wiry constitution which is able to resist fatigue and infection"

"Luckiness . . . pure luck is one of the chief factors making for happiness and success. Some men owe all or nearly all their success to luck, but if you have it not, remember that hard work and patience can make up to a great extent for the want of it"

"Brains . . . but not too many. It is unnecessary—perhaps dangerous—in medicine to be too clever. But if I had not many brains to bestow I should make up for it by an extra gift of diligence."

"Equanimity . . . there is no quality of mind more essential to you as doctors, for you will often

have to face sudden and disconcerting emergencies and a fair share of it will also do much to preserve you from the corroding effect of those worries which are unescapable in practice."

"A sense of justice in the first place to your patient—Justice also to your professional brethren—and lastly a sense of what is just to yourself."

"A sense of beauty . . . Disease is ugly . . . You will need a sense of beauty as a compensation and a way of escape, as a sanitizing and steadying influence."

"My last and best gift would be a sense of humor . . . It will help you to bear with the vagaries of your patients and still more with those of their relations and to derive amusement instead of annoyance from the eccentricities of your colleagues"

. . . "Armed especially with the last three, a sense of justice, of beauty and of humor, you will be able to face with equanimity all the buffets and disappointments, all the weariness and ugliness which your lives as doctors may have in store for you"

REFERENCES

- Bick, Edgar M. Source Book of Orthopaedics, ed 2, Philadelphia, 1948, W. B. Saunders Company.
- Billings, John W. History and Literature of Surgery. Dennis, F. S. System of Surgery, Philadelphia, 1895, Lea & Febiger, vol 1, pp 17-144.
- Fleming, A.: On Antibacterial Action of Cultures of Penicillium, Brit J. Exper Path. 10: 226-236, 1929.
- Hutchison, R.: Seven Gifts, Lancet 2: 61-62, 1938.
- Leonardo, Richard A.: History of Surgery, New York, 1943, Froben Press, Inc.
- Mettler, C. C.: History of Medicine, Philadelphia, 1948, The Blakiston Company.

but other *rarer sources* such as injection or ingestion of the material, not necessarily excluded from this type response. All soluble proteins of a certain molecular size, which are not naturally present in the body itself or that of a species closely related genetically, are antigenic, i.e., capable of producing the reaction. Antibodies, which are believed formed by the reticulo-endothelial cells and quite probably the mononuclear leukocytes, are globulin molecules modified to enter into physicochemical combination with the foreign protein. They are extremely specific and are demonstrated as bacteriolysins, preins, agglutinins, opsonins, antitoxins, etc., according to the result of their union with antigenic substance. Nonspecific complement is necessary in some cases to make the combination effective.

While immunological reactions are to a large degree beneficial and protective to the body, they may at times be quite the reverse and even fatal. Certain antibodies formed in excess become attached to the cells of the body and render them vulnerable to a subsequent union with antigen. This constitutes a form of hypersensitivity which is referred to as *anaphylaxis*. It would appear that the sensitive cells are destroyed by the antigen-antibody reaction with liberation of histamine or a histamine-like substance. The shock, which follows, manifests itself quite differently in different species, but in man is characterized by a fall in blood pressure, tachycardia, dyspnea, urticaria, and sometimes marked edema of the entire body. As the amount of antigen to produce shock must be relatively large and reach the tissues rapidly, anaphylaxis is encountered only experimentally, except in intravenous or intrathecal injections of immune serum in previously sensitized individuals.

The term *allergy* is applied more particularly to a form of sensitivity that develops naturally in certain individuals to some forms of foreign proteins. It implies an unusual responsiveness to specific antigenic mate-

rials derived from external sources. However, in a broader sense because of the similarity of the process, one may speak of a hypersensitivity to a particular infecting organism as bacterial allergy. This latter form no doubt plays a very great role in the character of the individual's response under such conditions. Here again antibodies have become attached to the cells of the body, but certain organs show a much higher degree of antigen-antibody reaction than others. For consideration of shock as a reaction to injury refer to Chapter IV.

Recently, more particularly through the investigative work on animals by Selye, attention has been directed to an integrated syndrome of closely related adaptive reactions of the body to nonspecific injury. This has been termed the *General Adaptation Syndrome* and is divided by its stages of development into the *Alarm Reaction*, the *Stage of Resistance* and the *Stage of Exhaustion*. The nature of the injury is not an important factor provided it is sufficiently severe. It may be external as a local trauma or burns or it may be internal as tissue destruction during disease or even initiated by mental stress. If it is of adequate intensity, the local peripheral nervous system sends stimuli which alarm the autonomic nervous system. The stimuli are amplified in the ganglia and passed on through the sympathetic system to the medulla of the adrenal from which large amounts of epinephrine and norepinephrine are secreted. This raises the blood pressure, mobilizes liver glycogen to provide glucose, dilates the pupils, and stimulates the anterior pituitary to secrete the adrenocorticotrophic hormone (ACTH) and the somatotrophic hormone (STH). The body is thus rendered alert.

With the activation of the adrenal cortex by ACTH to produce the gluco-corticoids (such as cortisone) and by STH, the mineralo-corticoids (such as desoxycorticosterone), the *Stage of Resistance* begins. These hormones in many respects appear to antagonize each other. The former causes involu-

the specific response of the individual. Probably no two persons will react in exactly the same manner to identical irritants. However, certain common types of response stand out for consideration and these may be divided into those which are *systemic*, or involving the body as a whole, and those which are more or less *localized* to the region of the site of injury.

SYSTEMIC REACTIONS

Systemic reactions occur more particularly in the presence of profound organic disturbance, generalized infections and severe trauma or stress. There is usually a certain amount of toxemia, and the more important reactions are fever, alterations in the white blood cells, increase in the sedimentation velocity of the erythrocytes, immunological reactions including anaphylaxis and allergy, shock, and the changes designated by Selye as the "alarm reaction" and "general adaptation syndrome."

Toxemia results from the absorption into the circulation of injurious agents such as products of tissue destruction and toxins from bacteria, viruses, etc. This may lead to fever, rapid pulse, general malaise, anorexia and, in children more particularly, delirium and convulsions. The rise in body temperature undoubtedly has a complex pathogenesis. It is believed due by some to the following course of events. The circulating toxic products act upon peripheral tissues causing protoplasmic changes in the cells, recognized as parenchymatous and fatty degeneration, with withdrawal of fluid from the blood and fall in blood volume. Drainage of blood from the vascular bed of the skin calls forth a reflex response from the heat regulating mechanism in the hypothalamus proper, with shivering and chills. Heat production within the body rises, but through the effect of the toxins on the controlling center, the body thermostat is set at a higher level and does not respond with a return of vasodilation of the skin and sweating until that level is reached.

Important information may be derived from the extraordinary differences which occur in the actual number of the various *white blood cells in the circulation* in response to injurious agents. In pyogenic infections due to the staphylococcus and streptococcus, and in pneumonia, appendicitis, etc., the increase in neutrophils is characteristic, while viruses tend to produce a neutropenia with increase in the mononuclear forms. Parasites and allergic conditions lead to an eosinophilia. The mechanism of this selective stimulation of the hematopoietic system is not very clear, but a pseudoglobulin, termed leukocytosis-promoting factor (L.P.F.), has been obtained from inflammatory tissue detritus. This causes the discharge of neutrophils from the bone marrow and stimulates leukopoiesis in the myeloid tissue.

An increase in the *sedimentation velocity of the erythrocytes*, known as the sedimentation rate, is of considerable diagnostic value in the recognition of an organic disturbance in the body. It occurs in practically all conditions where there is a breakdown of tissue or foreign protein enters the blood stream. The rate is therefore increased in toxemia and nearly all types of infection, also following the injection of vaccines or other foreign proteins, after severe trauma, fracture and operation, and in the presence of cancer if associated with destruction of tissue or inflammation. It is due to an increase in the positive electric charge of the plasma, which causes the negatively charged erythrocytes to clump into aggregates. Globulin tends to accelerate and albumin retard the velocity and the actual rate is apparently the result of a number of complex factors. Since the number of erythrocytes in the blood has a profound effect on the rate, a correction should be made when anemia is present.

Immunological processes include the systemic antibody reactions due to a foreign protein or complex polysaccharide, referred to as antigen. For the most part such foreign material comes from invading organ-

is in which whole masses of liver parenchyma are rapidly destroyed with shrinkage of the organ to half its normal size. These types of inflammatory response are classified *degenerative*. In such cases little is known of the detailed pathogenesis of the cell destruction. It has been attributed to toxins but this term is too broad and indefinite to be satisfactory. There is no doubt that the cells are subjected to a very profound and rapid alteration in their environment to which they are quite incapable of adjusting themselves, so that at first parenchymal, then typically degenerative changes take place in their cytoplasm and this may lead to necrosis. However, we are uncertain in many cases as to whether the physicochemical changes around the cells are brought about through the direct action of a noxious substance, or secondary to interference with the blood supply to the area as maintained by Ricker.

In the more frequent *acute* inflammations, or the most part due to bacterial infections with pyogenic cocci, the histopathological changes are principally vascular and *exudative*. They are well represented by the common boil. Here a chain of events develops, the links of which form a most interesting sequence and should be considered in some detail.

There first occurs a dilatation of the vascular bed with opening up of closed capillaries and acceleration of blood flow. This brings redness and blood heat to the part. It is quite definitely established that this is due to destruction of the body tissues either by trauma or the action of the bacterial toxins. This destruction leads to liberation of histamine or a similarly acting substance which by action on the neighboring cells and by a local axon reflex causes dilatation of the vascular bed. Menkin has isolated from injured tissue a polypeptide, leukotaxine, which he considers the immediate agent.

This acceleration is transient, however, and a slowing of the blood stream soon takes place. It is attributed to several factors in-

cluding the marked increase in volume of the capillary bed, swelling of the cells lining them, and transudation of fluid raising the viscosity of the blood. The axial stream is lost and the lighter leukocytes go to the edge where they eventually become attached to the now irregular lining. Emigration of the polymorphs takes place apparently through the cytoplasm of the endothelium by pseudopodial activity. They are attracted by the positive chemotactic influence of the tissue detritus. Erythrocytes may follow but play only a passive role. The content of the exuded fluid varies with the intensity of the reaction from that of low protein content to almost whole blood plasma with fibrinogen which clots to form fibrinous threads. The high phagocytic activity of the neutrophil polymorphs plays a beneficial role in engulfing bacteria and particles of cell detritus, moreover these cells disintegrate with the liberation of proteolytic ferments. On the other hand, certain foreign proteins, parasites, and allergic conditions seem to produce a specific chemotactic influence on the eosinophilic white blood cells.

With the attenuation of such an acute reaction and in the *subacute* forms of inflammation, the changes characterized by a *proliferation of the fixed tissue elements* play the important role. They contribute the bulk of the mononuclear forms in the area though some may arise through diapedesis from the blood stream. The ubiquitous root cells of the reticulo-endothelial system multiply and differentiate into fixed and wandering histiocytes which form the highly phagocytic macrophages, scavengers of cell detritus. Foci of lymphocytes appear and by the disintegration of these mononuclear forms valuable immunological products are in all probability brought into the field. Proliferation of fibroblasts and endothelial cells occur and this may result in the formation of a dense membrane that blocks the spread of an infection. Such changes are well exemplified in the wall of an inflamed gall bladder and in the lower grade types of appendicitis.

tion of the lymphatic tissues and thymus and elicits characteristic changes in the white blood cells, while the latter profoundly affects the cardiovascular system, the blood pressure, and the kidneys. These hormones, in contrast to the initial phase where carbohydrates were made immediately available and there was a loss of electrolytes and water from body fluids, increase the utilization of fat, either directly or by conversion to liver glycogen, and through action on the renal tubules conserve sodium and water. The changes produced by medullary activity of the adrenal are thus balanced by cortical activity and the individual is in a position to deal more satisfactorily with the period of stress.

Although the body may come to adapt itself to repeated injuries of identical nature so that it handles them more adequately, if too severe, a *Stage of Exhaustion* develops with collapse of the protecting mechanism. This expresses itself as involution of the thymicolymphatic system, loss of adrenal lipids, gastrointestinal ulcers, etc.

LOCAL REACTIONS

In contrast to the generalized processes outlined above, the local reactions of the body to injury are referred to as *inflammation*. There is considerable difference of opinion, however, as to the proper usage and precise definition of this term. The surgeon does not, as a rule, speak of a wound as inflamed, so long as it is clean and has not become septic, though local reaction is present. Moreover, some pathologists include under inflammation only that part of the local changes which is of exudative character, while all proliferation of cells and fibrosis taking place at the site of the injury to them constitute repair. These difficulties and differences arise from a didactic and teleological approach to the subject. The cells of the body at the point of injury undergo regressive and progressive changes purely as the result of their natural reaction to alterations

in their environment. These individual cell responses may or may not be beneficial to the body as a whole. Very different combinations of these cell reactions are met with in the various types of inflammation, and many of the same cell changes are encountered in repair. To assume that the fibroblasts proliferate in one case to check the spread of an infection and in another to replace damaged tissue is drawing a purposeful distinction. Consequently any attempt to sharply define inflammation and repair as separate processes must of necessity fall down on natural grounds.

The classical signs of inflammation calor, rubor, dolor, tumor and functio laesa are explained as follows. Following a brief period of vasoconstriction which terminates with the paralysis of the vascular bed, the larger vessels dilate and there is an increased flow of blood through the affected area. A distinct flush occurs and with this a feeling of warmth. The temperature of the part has been raised by the hyperemia in that of the deeper structures, but not above it. Extravasation of fluid at first leads to swelling, which pits on pressure, but as the exudate becomes cellular the tissue is much firmer. Pain is present provided there are pressure-receptor nerve endings in the area. The loss of function is also a very variable attribute depending on how greatly these changes interfere with the activity of the particular part involved.

Inflammatory reactions may be divided according to the duration of their course into acute, subacute and chronic. This, however, is primarily a clinical classification, and expresses in no way the vast differences in the character of the histopathological changes met with at the site of the injury. In the fulminating conditions, the lesion is destructive with marked degeneration of the parenchymal cells, and also sometimes the more resistant reticular, vascular and fibrous tissue. This goes on to necrosis. Exudative changes are less pronounced. Such a lesion is well exemplified by fatal cases of infectious hepa-

for example in the tubercle which produces its epithelioid and giant cells through the proliferation of fixed histiocytes and then it enlarges the center undergoes a caseous crisis. Sometimes, as in actinomycosis, purulent foci of exudate appear.

We meet, therefore, in the various forms of inflammatory reactions, different combinations of local tissue changes that may be predominantly degenerative, exudative, proliferative, or productive, depending primarily on the character of the irritant, but in which any other factors contributed by the host play a modifying role. If the action of the irritant in an area ceases, which in the case of simple trauma is early, but with infection may be only after a very long drawn-out process, replacement of the damaged tissues takes place with scar formation. This does not mean, however, that in all inflammatory processes only the exudative, and not the proliferative and productive changes should be included as part of the inflammatory reaction.

REPAIR

Repair is the process of restitution to integrity of the site of an injury. Complete and unrecognizable return of the area to its former state rarely occurs, and the degree to which this is accomplished is influenced by a large number of factors, both local and general. The process is for the most part one of cell multiplication with maturation. This may be due to certain growth-promoting chemical substances derived from the cells of the damaged tissues and lymphocytes. These have been called *trephones*. It seems, however, rather to rest in an upset in the equilibrium of the cells brought about by the injury and the inflammatory changes associated with it. These alter the environment of the cells with loss of the normal controlling inhibitive influences so that multiplication is allowed to proceed until equilibrium is restored.

As a general rule it may be stated that the more highly differentiated and specialized

cells such as nerve and parenchyma, regenerate less readily than stromal elements; however, the surface epithelium of the skin, bowel, or respiratory tract, which normally is in proliferative activity retains this ability. Maintenance of the framework of the organ at the site of the injury is of great importance for regeneration of parenchymal cells. If this is preserved they will multiply in orderly arrangements as, for example, the columns of the liver in infectious hepatitis and the kidney tubules following mercury poisoning. However, where the inflammatory changes have been severe and the scaffold destroyed, fibrous connective tissue fills in the gap with eventual scar formation. This indicates the dictating importance of the influence of cells on one another in the process. The maintenance of a good blood supply and adequate lymphatic drainage is essential for rapid healing. The chronic circulatory ulcers of the leg exemplify what happens when such is wanting. The surgeon should guard against any mechanical restriction to arterial flow to the affected part. Recently more attention has been directed to general biological factors that play an important role in repair. Vitamin deficiencies, more particularly vitamin C, are a distinct impediment and the need of an adequate supply of proteins for fibroblastic proliferation is recognized. The patient should be brought, as soon as possible, by high protein diet into positive nitrogen balance.

When union of severed tissues is brought about without the necessity of filling in any appreciable gap it is referred to as *healing by first intention*. This is well exemplified by the clean incised wound of an operation. The approximated edges are united by a thin layer of fibrin coagulum derived from the extravasated plasma and lymph. Within three days, proliferating fibroblasts and endothelial buds have grown into the mesh from each side and eventually unite. Phagocytic blood and tissue cells clear the tissue debris. Maturation of the connective tissue elements with laying down of reticular and collagen

Because of the vast number of factors involved, such inflammatory processes vary considerably in their course and not infrequently develop particular features which have come to be recognized as *pathological entities*. The term *pus* is applied to a semifluid mixture of inflammatory exudate, predominately polymorphonuclear, with necrotic cell detritus and bacteria if they constitute the etiological factor. In solid tissue a collection of pus usually tends to localize with the formation of an *abscess*. Its spread may be restrained by a surrounding zone of inflammatory granulation tissue, known as a *pyogenic membrane*, but an abscess will not readily heal unless it spontaneously evacuates itself by extension to a surface or is surgically incised. In cases where the pus does not localize but spreads in sheets and strings throughout the surrounding area, the rather inappropriate term *cellulitis* is applied. This occurs more particularly in streptococcal infections and in loose areolar tissue. If such an inflammatory process is associated with considerable necrosis of tissue, it is spoken of as *phlegmonous*. Pus that has accumulated in a pre-existing cavity, often with its enlargement, is called an *empyema*. This condition is met with commonly in the pleura, gall bladder, and appendix.

Inflammatory processes due to infections often spread by following the lines of least resistance along tissue planes and eventually "point" with rupture on to the surface forming a *sinus*. The tract is lined by a zone of inflammatory tissue which may bear the distinguishing features of the etiological agent, as in tuberculosis and syphilis. Should the tract result in communication between two different surfaces a *fistula* is formed.

An *ulcer* may be defined as an interruption in the surface continuity of an organ with accompanying inflammation. The skin or mucous membrane is most frequently involved. The term does not include superficial erosions or abrasions which heal by simple regeneration. There is, however, a vast difference in the speed of healing by

granulation tissue of the so-called healthy ulcer and some chronic indolent lesions such as the rodent ulcer, which actually tends to increase in size because the destructive activity of the neoplastic process persists.

Necrosis is the local death of cells, that is of tissue *en masse*. The term, as employed surgically, does not apply to the death of isolated cell elements or of the body as a whole. It occurs whenever the tissues in an area can no longer adapt themselves to the profound environmental alteration. The causes are innumerable but all belong fundamentally to either the chemical or physical group. The histological appearance of the necrotic tissue varies with the degenerative changes which took place prior to the necrosis and the alterations, mostly enzymatic, after death of the cells occurred. One may distinguish simple, coagulative, colliquative and caseous forms. If putrefaction develops in the necrosed area, *gangrene* results. This is most common in the leg due to obstruction of the blood supply, but it should be borne in mind that the so-called "dry gangrene" without putrefaction is strictly speaking only an ischemic necrosis.

The *chronic* types of inflammation may be divided into nonspecific and specific. The former are for the most part without a clearly understood etiological factor, while in the latter the cause is known and they possess a definite histopathological stamp referred to as *granulomatous*. Tuberculosis and the gumma of syphilis are the most common granulomatous forms. In these slowly progressing local reactions to an irritant, vascular hyperemia and exudation are for the most part minimal. It is proliferation of the cellular elements of the stroma and productive changes, by which is meant the laying down of collagenous fibrous tissue, that control the histological picture. Parenchymal cells are frequently destroyed and may show attempts at regeneration. Endothelial budding with the formation of granulation tissue builds up the bulk of the lesion. Necrosis of the newly formed areas of reaction are not uncommon

periostrum which has thickened due to proliferation of its fixed tissue and blood vessels and also from the fibroblastic and endothelial elements of the bone. It is completed thin a few days and is referred to as the *callus*. During the following week consolidation of this soft granulation tissue occurs with transformation into dense collagenous connective tissue, in which degrees of transformation to cartilage take place. This is the *fibrocartilaginous callus*. To complete the bony union it must be replaced by new bone which grows in from the peripheral periosteum and endosteum in the same manner as endochondral ossification. Under favorable conditions this calcifies as laid down, and when the gap is bridged the *bony callus* is complete. Subsequent reorganization to meet the lines of stress is a process of many months' duration. It requires the destruction of portions of the newly formed bone by osteoclastic activity and the laying down of lamellae of new bone where it is needed by osteoblasts.

Both general and local factors influence the healing of bone but the latter are by far the most important. It is extraordinary what excellent repair may occur in cases suffering from profound debility. Adequate supplies of protein and calcium are necessary and the effects of Vitamin C and the parathyroid hormones on calcium metabolism must be borne in mind. Locally a proper blood supply to the area is imperative for satisfactory healing. Should this fail there is delay in the removal of cell detritus and a persistence of acidity in the tissues. This interferes with the deposition of calcium that can occur properly only with a return of the local fluids to an alkaline pH. *Nonunion* results with sclerosing fibrous and uncalcified, osteoid tissues. On the other hand hyperemia stimulates resorption of bone which may be out of proportion to the amount of deposited callus, and *delayed union* results. Of course, many other factors such as the approximation of the parts, infection, etc., play an important role.

The endothelial cells lining *serous cavities* such as the pleura and peritoneum proliferate readily and resurface any denuded area. However, if a fibrinous exudate has occurred and adjacent areas become glued together, the exudate is organized by fibrous tissue forming an adhesion, and then the endothelial cells grow over the band, surfacing it. This tends to explain its persistence and long preservation. Regeneration of the skin is fully described under Burns.

In the strict sense *compensatory hyperplasia* must be distinguished from the processes of repair though it also follows injury. It is concerned with a multiplication of the cells of an organ to make up functionally for those which have been destroyed. If one kidney is removed the other enlarges and this is due to lengthening of the nephrons. After a portion of the liver is excised a diffuse hyperplasia takes place throughout the remaining parenchyma until the bulk of the functioning tissue is replaced. The myeloid metaplasia of fatty bone marrow in persistent blood loss is a similar reaction. We have very little knowledge of the factors concerned in bringing about such compensatory adjustment.

There remains to be considered certain, fortunately uncommon, conditions in which an excessive *overactivity of the repair process* occurs. In this it would appear that the usual restoration of a state of equilibrium in the tissues with completion of the healing process does not take place. In most cases this is on the part of the proliferating fibrous connective tissue, but some epithelial growths seem to fall in this category.

In the Negro race, more particularly, the fibrous tissue in a wound of the skin may not cease to proliferate, but continues to form dense collagenous tissue so that a hard thickening is produced. This so-called *keloid* may follow very trivial injury, and as it is likely to recur if excised, would appear to depend on some unexplained peculiarity of the individual. Following trauma of the abdominal wall, either from childbirth injury

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Should there be a gap necessitating the filling in by granulation tissue, the process becomes *healing by second intention*. This occurs where the edges cannot be brought together, in ulcers and when infection with destruction of tissue or extravasation of blood complicates the repair. There is no fundamental difference between the two types of healing, but the second takes much longer; disproportion in the proliferative activity of the various tissues as in proud flesh is common and the eventual scar is permanent. Small extravasations of blood in tissues referred to as ecchymosis can be completely resolved by lymphatic drainage and phagocytosis of the products of erythrocytic disintegration. A hematoma, however, requires organization by granulation tissue. Removal of the large bulk of blood pigment and lipoids may be difficult and products frequently remain behind in the scar, or the whole becomes an encysted mass of fluid blood detritus.

The degree of actual regeneration varies greatly in the different specialized types of tissue. For this reason some require further consideration. In the *nervous system* the neuroglia and cells of Schwann take the place of fibrous connective tissue as the readily proliferating elements in repair. Damage to the nerve cells and tracts of the brain and cord is irreparable, but if a peripheral nerve is severed at a point not too close to the cell body, regeneration is possible. Mild degenerative changes take place in the cell and in the medullary sheath of the proximal axon near the point of injury while the whole of the distal segment undergoes an only Wallerian degeneration. The Schwann cells proliferate at the site and along the sheath, and into this neurolemmal tube sprouts from the proximal axon make their way, eventually attaching themselves to motor or sensory end organs with partial restoration of function. This is, however, a comparatively slow proc-

ess. Under circumstances such as an amputated stump, where the possibility of union between the two parts has been removed, a jumbled mass of regenerated Schwann cells, fibroblasts and axonal sprouts combine to form the so-called *amputation neuroma*.

The regenerative activities of the various tissues of the *locomotor system* differ considerably. *Striped muscle* is usually replaced by fibrous tissue. However, following injury, peculiar multinucleated bulbous sprouts do develop at the ends of the fibers and these may later become striated. Apparently, if the sarcolemmal sheaths are preserved, new fibers will grow into them with good restitution of the structure. The same lack of active regeneration holds for cardiac and smooth muscle although the bowel wall often fails to show any appreciable scarring of the muscle layer when examined histologically some time after an end-to-end anastomosis. In spite of the fact that *cartilaginous tissue* is so readily formed in the callus of a broken bone, adult cartilage does not readily regenerate if fractured or incised, but is united by fibrous tissue. *Tendon*, on the other hand, is well repaired particularly if the sheath is preserved. The coagulum between the approximated ends is organized by collagenous fibrous tissue which is then orientated by the lines of stress into dense bundles. Lengthening is likely to occur if the gap is large.

In the repair of *bone*, exemplified by the healing of a simple fracture, the process is somewhat more complex, though it follows the same general principles. Actually bony tissue itself has very little regenerative ability and has to depend upon its supporting and vascular elements for such activity. Following the break, due to rupture of blood vessels and tearing of soft tissues, a coagulum of blood and tissue detritus is formed. This fills the gap and pushes outward the periosteum where it is stripped off the fragments. Organization of this coagulum then takes place in the usual manner by the ingrowth of granulation tissue. This takes origin from

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CHAPTER III

SURGICAL BACTERIOLOGY AND CHEMOTHERAPY

GERTRUDE G. KALZ, M.D.

The purpose of this chapter is to give the student a general survey of the infections he is most likely to encounter in surgical practice. No attempt is made to give a complete list of all the infections which may occur or to discuss details of bacteriological technique or of the physiological and other characters of the bacteria. The microbes will be discussed only in their relation to disease and only those characteristics will be selected for notice which are essential for the understanding of the disease process and for a rational approach to treatment with either antisera or modern chemotherapeutics. It is obviously impossible to make a strict division into surgical, medical or other infections, but certain types are by their very nature treated by the surgeon, whereas others, although they may occur in a surgical patient, are by convention treated by the physician. No consideration will be given to the latter type of infection in this chapter.

Surgery owes a great debt to Lord Lister (1827-1912) who was the first to realize the importance of Pasteur's work for the field of surgery and made an important contribution by applying the knowledge of his time to the operating room. Modern asepsis is only a further development of those first important concepts of antiseptis. Due consideration is given to the whole problem of sterilization and asepsis in the chapter on Surgical Technique.

HOST-PARASITE RELATIONSHIP

The host-parasite relationship involves certain structural and physiological features of the bacteria which play a part in their adaptation to parasitism and the responses of the invaded host to the bacteria and their

products. To evaluate the risks of infection in the operating field the surgeon should recognize the natural presence of certain kinds of bacteria in the various parts of the body, as well as possible outside sources. He must, therefore, be familiar with the means of preventing bacteria from entering wounds and of treating already established infections.

Pathogenicity is the broad term used to express the concerted action of a number of different characteristics which together enable microorganisms to cause disease. The type of disease is determined by the specific organism and its products but the responses of the individual host give character of clinical importance to the disease. A variety of factors in the host are of great individual and collective importance and determine the degree of susceptibility and resistance to the infecting organism. Other features, like the site of localization or the organ or system invaded are also of great significance. Certain bacteria have become adapted in certain localities and are accepted by the body without eliciting any reaction and cannot be completely eliminated from the region. Thus, the flora of the intestinal tract, of the mucous membranes, and of the skin are examples of a well-balanced state between host and parasite, which is not only harmless, but as in the case of the intestinal flora may even be beneficial in synthesizing essential substances (vitamin K, and some of the B Complex). However, if some of these bacteria stray from their usual site into different tissues or organs they may and do cause disease, although, not all bacteria found in and about the body are capable of doing this. In order to cause disease, it is usually essential for any pathogenic organism to

gain access to the body tissues and to be able to multiply there to a sufficient degree. The mechanism by which specific organisms cause disease varies; some cause their effects by powerful toxins and/or enzymes absorbed from a site of localization (diphtheria, tetanus) and in some cases produced outside the body (botulism), others produce disease by active multiplication and local injury at the site or sites of invasion (staphylococcus abscess, impetigo). A third group of bacteria may combine both modes of attack (gas gangrene) and yet others characteristically cause rapidly generalized infection and septicemias (typhoid, syphilis). Whether or not an infection remains localized or becomes generalized depends to some extent upon the bacterial species involved and upon the defense mechanism of the host and various degrees and characters of infection may be seen in different cases due to the same species of bacteria (*M. tuberculosis*, streptococci, *B. anthracis*). Certain bacteria show selective localization in particular organs or tissues.

DEFENSE MECHANISM OF HOST

The response of the host to the microbe is manifold depending in part on the particular species of microbe and in part on several factors in the host itself. We have said above that a prime factor in the establishment of an infection is the successful penetration by the bacteria or their products into the tissues of the host. The natural barriers which man possesses have to be broken down before access can be obtained. The normal self-sterilizing ability of healthy skin is a very successful line of defense but even in the unbroken skin there are weak spots which the enemy can attack: the hair follicles and the ducts of sebaceous glands represent such openings, and the bacteria, particularly *Staphylococcus*, may take advantage. Certain bacteria, such as *Pasteurella* and *Leptospira*, are thought to be able to penetrate uninjured skin and mucous membranes. Wounds or other injuries to the skin provide a point of attack for a va-

riety of bacteria. The portal of entry for the respiratory tract is the nose and nasopharynx. The mucous membranes, phagocytes, and the various secretions of the nose, pharynx and the mouth provide a fairly effective barrier. Lysozyme, a bacteriolytic enzyme occurring in nasal secretions, tears and other body secretions may afford some protection against certain bacteria and a certain degree of resistance may be provided by antibodies in the mucosal secretions. If any of these natural defenses are altered, the penetration of pathogenic bacteria is facilitated and infection of the upper respiratory tract will occur. The direct penetration of airborne particles to the pulmonary alveoli is difficult, but the inspiration of infected materials and secretions may cause serious trouble.

Infections of the intestinal tract occur after the bacteria have passed several defense hurdles. In the mouth, the saliva has an inhibitory action for some. The highly acid gastric juice will destroy another number of organisms, but many do pass this barrier in food which may act as a buffer or pass rapidly through the stomach in a fluid medium, and still others (*tubercle bacillus*) can survive for a certain length of time in the gastric juice. The type of flora found in the small and large intestine is determined by the anatomical structure, the secretions of the mucosa and accessory glands, and the dietary habits of the individual. Any upset in the normal physiological conditions may permit the establishment of a number of intestinal pathogens.

In the healthy adult female the acid pH developing in the secretion and the type of epithelium of the vagina prevent fairly effectively the implantation of particular bacteria, e.g., *coliforms*, and may also restrict to some extent localization of gonococci, which can more easily establish themselves in the vagina of prepuberty children.

If bacteria penetrate these outer barriers, a second line of defense is provided by the process of phagocytosis and by "natural

antibodies." Phagocytosis represents an attempt of the body to rid itself of extrinsic matter and is highly successful with non-pathogenic organisms which reach the blood stream or tissues.

If pathogenic organisms are introduced into the blood stream, the same mechanism is mobilized by the host but the clearing is less successful because the ability to resist phagocytosis is a fairly common characteristic of virulent bacteria in general and with some bacterial species it is the chief attribute of their pathogenicity. Even if pathogenic bacteria are taken up by phagocytic cells and a number of them are destroyed, the majority will escape after injuring or even killing the cells and start their process of multiplication or adaptation in various tissues. Most bacteria are not directly introduced into the blood stream but rather into some tissues or organs and the immediate reaction of the body defences is to localize and fix the injurious agent; the first clinical manifestation of the interaction between host and parasite is local inflammation. Polymorphonuclear leukocytes are mobilized from the blood stream and accumulate locally. After this initial wave of polymorphonuclear cells, which may be very brief in some infections, a second type of cells, the mononuclear macrophages make their appearance and increase in numbers. Depending upon the bacterial species and its virulence this mechanism is more or less effective. If it proves insufficient to destroy the majority of organisms, an invasion of lymph channels and blood vessels takes place and generalized infection will follow. This cellular clearing mechanism is augmented and supported by components of the serum. It has been known for a long time that the sera of man and different animal species contain antibodies against various bacteria and red blood cells of other species. These antibodies which can render bacteria more susceptible to phagocytosis, or agglutinate or lyse microbes are connected with the serum globulin. They are often called

"natural" or "nonspecific" or "physiological" antibodies because they are present in the serum of man or animals who are not known to have experienced previous infection with a given organism. The origin of these "natural" antibodies is still a hypothetical question, though they are more common and more abundant in the course of epidemics and are attributed by many to subclinical infections by others to common antigens. The hemagglutinins that divide human blood into different blood groups are special examples of natural antibodies. The exact role of the "natural" antibodies in resistance to infection is not clearly established. Contrary to these antibacterial antibodies whose origin and exact value are still debatable, so-called "naturally" occurring antitoxins are almost certainly due to subclinical infection with the respective toxin-producing organisms and they do confer antitoxic immunity of a specific type if present in sufficient amounts. This has been proved for diphtheria, tetanus, and scarlet fever. Of greater importance as defensive forces against infection are the antibodies which are formed in answer to a specific stimulus with the object of removing and neutralizing the bacterial or toxic substances or both. These antibodies are named "specific" or "immune" antibodies and they too are connected with the serum globulins. These antibodies are specific for the particular bacteria or their antigenic components; for instance antibody produced against *Pneumococcus* Type I will only combine with Type I polysaccharide and is not effective against Type II, or antibody against *Haemophilus* Type II will only act on Type II and not on Type A, F, etc. Immune antibodies show a rising titer in recovery from an infection and can be transferred from man to man or animal to man and furnish protection against specific infection.

The formation and specificity of antibodies is one of the most fascinating subjects in biology. The site of formation of these antibodies is still a controversial subject and cannot be discussed in this connec-

tion. Up to the relatively recent introduction of chemical agents into the treatment of infections, antibodies either actively produced or mainly passively introduced were the most important weapon in the fight against pathogenic microorganisms. Even in this era of antibiotics, antitoxins or antibacterial sera are still a valuable aid in the treatment of disease, and antitoxin is still pre-eminent in diphtheria and essential in tetanus. Apart from their importance as therapeutic agents, the appearance of specific antibodies in response to certain infections is of great significance in the diagnosis of infectious diseases, as is illustrated by the Widal reaction, the Wassermann reaction, Brucella agglutination, and a series of other similar serological tests, all based upon the demonstration of antibodies against specific microorganisms. The studies on the mechanism of antigen and antibody reactions have been tremendously stimulating and useful for decades and have led to a series of new concepts in immunity. The applications of highly specific antigen-antibody reactions have also resulted in the recognition of a variety of components in the bacterial cell which in turn has led to more exact identification of bacterial species, to new tools in the field of epidemiology and to better appreciation of the processes of infection. An understanding of immunological principles is of fundamental importance in every branch of medicine, for diagnostic, preventive, and curative purposes.

The interaction between host and parasite in all its complexity determines the clinical course of an infection as well as the pathology of the lesions. The clinical signs and symptoms and the surgical treatment of various infectious processes are discussed in the appropriate chapters of this textbook. The description of tissue responses to bacterial invasion in general (inflammation, necrosis, fibrosis, etc.) or to certain specific microorganisms, e.g., tubercle bacillus, *Treponema pallidum*, fungi, is subject matter of pathology, and their discussion is omitted with the

assumption that the student is thoroughly familiar with all the aspects. But, although perhaps redundant, a brief definition of terms which are commonly used in connection with infections is appended to prevent confusion of interpretation.

Abscess is a localized necrosis with a collection of pus confined by a wall of granulation tissue and may be acute or chronic. The furuncle or boil is a common form of abscess of the skin caused by *Staphylococcus pyogenes* which usually gains entrance through the hair follicle.

Cellulitis is a diffuse, often rapidly spreading inflammation of the subcutaneous tissues, frequently caused by the hemolytic streptococcus and involving the fascia. A serosanguineous exudate in which leukocytes are sparse is rather distinctive.

Bacteremia means the simple presence of bacteria in the blood stream, which may be transitory. This is a relatively frequent occurrence after tooth extraction or tonsillectomy without causing any serious symptoms.

Septicemia is a bacteremia with severe generalized symptoms often accompanied by valvular lesions and other localizations.

Septicopyemia indicates a septicemia with abscess formation in various organs, usually initiated by infected emboli. These abscesses are known as "pyemic abscesses."

Toxemia signifies that the clinical signs and symptoms are due to a toxin rather than directly to bacterial multiplication. The best example of a toxemia is tetanus.

GENERAL ASPECTS OF THE TREATMENT OF INFECTIONS

The development in the treatment of infectious diseases can be divided roughly into three phases. The landmark in the first phase, which might be termed the *antitoxin-antiserum phase*, was set by Roux and Martin (1894) who immunized horses with diphtheria toxins and produced antitoxin. The extension and elaboration of this principle to

other infections have been of immense value in the prevention and treatment of bacterial diseases. Behring and Wernicke (1892) actually treated for the first time a case of human diphtheria with serum from immunized animals. This marks the beginning of specific serum treatment. Infections in which treatment with antitoxin is still of prime importance will be discussed in the special part.

The second phase in the treatment of infections started with Ehrlich's work on chemotherapeutic agents (1904-1915) crowned by the discovery of "Compound 606" (1910), which as Salvarsan and all its related compounds has been the most effective weapon against syphilis for almost 35 years. The next important step in this phase was made with the introduction of Prontosil by Domagk (1935). The dramatic effect of Prontosil in streptococcal infections, particularly erysipelas, led to intensive investigations, and it was soon discovered by some French workers that the active principle of Prontosil was sulfanilamide. This compound was soon followed by more active and less toxic derivatives like sulfapyridine, sulfathiazole, sulfadiazine, sulfamerazine, Gantresin, and many others.

The usefulness of the sulfa drugs in surgery is somewhat limited by the fact that their activity is considerably decreased by the presence of pus and tissue debris in wounds. A further disadvantage of these drugs is their relative toxicity as compared to penicillin or other antibiotics, and that many species of bacteria are naturally resistant and others quite frequently develop a high degree of resistance after exposure to these drugs. On the other hand, some sulfa drugs penetrate more freely into the spinal fluid than penicillin and are still considered the drugs of choice in meningococcal meningitis, in certain urinary tract infections (*E. coli*), and in bacillary dysentery (*Shigella* infections). Sulfadiazine or one of the poorly absorbable sulfonamides (sulfaguandinine, sulfasuxidine, sulfathalidine, etc.),

either alone or in combination with oral streptomycin, is used in the preparation of the intestinal tract for gastrointestinal surgery. "Triple sulfas," a combination of three sulfa drugs in microcrystalline form, is preferred by many surgeons both for systemic and topical treatment; the incidence of precipitation in the renal tubules appears to be lower. In practice today, the sulfonamides are being more and more replaced by the various antibiotics in the treatment of infectious diseases.

The third and present phase in the treatment of infections, which logically is a continuation and part of the chemotherapeutic era, begins with the discovery of penicillin by Fleming (1929) and its development for clinical application by Florey and Chain and many others (1940). This period of antibiotics, although of such recent origin has had the most stupendous effect on the whole field of medicine and allied sciences. The ever increasing number of available antibiotic agents, all rather specific in their action, has put added importance on accurate diagnosis of bacterial infections, for the proper choice and use of the most effective agent. It should be recognized and appreciated that our knowledge in this field is still very limited and based to a large extent on empirical findings. It is therefore prone to rapid changes in concept and practice, and statements as to choice of drug and dosage are only valid for our present state of experience.

It cannot be emphasized too strongly that all these modern "miracle" drugs do not permit the surgeon the slightest deviation from accepted standards of aseptic technique. They do make possible in many instances successful operations under conditions which formerly were followed by a high mortality rate. Skillful surgery remains of utmost importance, and proper attention to nutrition, hydration, and all other supportive measures is most essential.

At present well over 150 antibiotics have been isolated from different microorganisms

and plants. Only relatively few of them, however, have been found useful clinically. In choosing an antibiotic or a combination of antibiotics for treatment, certain factors have to be considered

The Bacterial Spectrum of an Antibiotic.—This term is used to define the activity of a particular antibiotic against various microorganisms. We speak of a broad or narrow spectrum, depending on the range of inhibitory action against numerous or only relatively few species of microbes which an antibiotic may demonstrate. The selectivity of action is a distinctive character of antibiotic substances which distinguishes them somewhat from antiseptics which have a less obvious selective range.

Table I indicates the efficacy of the antibiotics and sulfonamides at present most widely used against the more common infectious agents

This schematic outline of the antibacterial spectra does not imply that a specific antibiotic is equally effective against all strains of a bacterial species. For example, not all strains of staphylococcus are equally susceptible to penicillin, and this species shows considerable differences in sensitivity. This consideration is particularly applicable to streptomycin, which exhibits great variation against different strains of the same species. A so-called sensitive strain must be inhibited by amounts of a given antibiotic which can be practically obtained and maintained in the body or in a particular localization. By the same definition a resistant organism is not necessarily completely unaffected by an antibiotic substance, "resistant" merely indicates that it is not inhibited by doses which are practicable for treatment

Apart from these prime problems of relative sensitivity or resistance of an organism causing infection, there are a number of other aspects which must be considered in the choice of an antibiotic, in the dosage used and in the mode of application. Wherever possible, the infecting organism should be isolated and its sensitivity determined. In

mixed infections due to gram-positive and gram-negative organisms, combined therapy should be used to avoid proliferation of one group over the other. Many gram-negative organisms produce penicillinase, which destroys penicillin, and if this antibiotic is used in such infections its value is questionable. For the treatment of systemic infections the concentrations of the chosen antibiotic should be maintained, at least during certain recurring periods, 5 to 10 times above the minimal inhibitory amounts for the particular organism. The distribution in the body varies somewhat with the different antibiotics.

Penicillin diffuses after parenteral introduction into most tissues, but does not penetrate freely into the bone marrow, the joints, body cavities, the intact central nervous tissue and not very readily into the spinal fluid even in cases of meningitis unless very high doses are used. In cases of empyema or other localized infections which are accessible to topical treatment, a combination of topical and systemic treatment is advisable. Antibiotics in the form of aerosols either alone or in combination with intramuscular injections, may be the treatment of choice in certain types of upper respiratory infections

Streptomycin, like penicillin, is readily absorbed in the blood stream after intramuscular injection and is distributed to the various tissues. On the whole, as far as dosage and route of administration are concerned, very much the same rules apply as for penicillin. But streptomycin has two aspects which deserve serious consideration, particularly if prolonged treatment is indicated. One factor is toxicity and the second is the development of resistance of initially sensitive organisms. Both factors are particularly important in the treatment of tuberculosis, but the development of resistance may occur very rapidly and is therefore on occasion a major problem in the treatment of acute infections. A combination of strep-

TREATMENT OF INFECTIONS

TABLE 1. SPECTRA OF THE IMPORTANT ANTIBIOTICS
Adapted From Scope, December, 1950

| ORGANISMS | PENICILLIN | STREPTOMYCIN | AUREOMYCIN | CHLORAMPHENICOL | NEOMYCIN | TERRAMYCIN | POLYMYXIN | BACITRACIN | TYROTHRICIN | REMARKS |
|---|------------|--------------|------------|-----------------|----------|------------|-----------|------------|-------------|--|
| Gram-positive | | | | | | | | | | |
| Staphylococcus pyogenes | 1 | 2 | 2 | 2 | 3 | 2 | 5 | *4 | 4 | Antitoxin of value in systemic infections |
| Streptococcus pyogenes | 1 | 2 | 2 | 2 | 5 | 2 | 0 | *4 | 4 | Sulfonamides also effective. |
| Streptococcus viridans | 1 | 2 | 2 | 5 | 5 | 2 | 0 | *4 | 4 | Combined treatment with pen & Streptomycin in s.b.c. |
| Enterococci | 2 | 2 | 1 | 5 | 5 | 2 | 0 | 5 | 5 | |
| Pneumococcus | 1 | 2 | 2 | 2 | 5 | 2 | *4 | *4 | 4 | Antitoxin. |
| C. Diphtheriae (as adjuvant to antitoxins only) | 1 | 5 | 5 | 5 | 5 | 5 | 0 | 5 | 5 | |
| B. Anthracis | 1 | 0 | 2 | 5 | 5 | 3 | 5 | 5 | 5 | Antitoxin essential in tetanus |
| Clostridia | 1 | 0 | 5 | 0 | | | | 5 | | |
| Actinomyces israeli | 1 | 0 | 2 | | | | | 5 | | High doses over prolonged period. |
| Gram-negative | | | | | | | | | | |
| Neisseria | 1 | 2 | 2 | 2 | 5 | 2 | 5 | *4 | 5 | Sulfonamides also effective. |
| E. coli | 3 | 2 | 1 | 1 | 3 | 2 | *4 | 0 | | Chloramphenicol in typhoid. Sulfonamides effective. |
| Aerobacter | 0 | 2 | 1 | 1 | 3 | 2 | *4 | 0 | | |
| Klebsiella | 5 | 1 | 1 | 1 | 3 | 3 | *4 | | | |
| Salmonella | 5 | 3 | 3 | 2 | 3 | 5 | 5 | 0 | | |
| Shigella | 0 | 2 | 2 | 1 | 3 | 5 | *4 | 0 | | |
| Proteus | 5 | 1 | 5 | 2 | 3 | 0 | 0 | 0 | 0 | Combined aureo. & strepto. |
| Pseudomonas | 0 | 1 | 2 | 3 | 3 | 2 | *4 | *4 | 0 | |
| Pasteurella (tularensis) | 0 | 1 | 1 | 2 | 5 | | 5 | 0 | | |
| Brucella | 0 | 2 | 1 | 1 | 3 | 2 | *4 | 0 | | |
| H. influenzae | 5 | 1 | 1 | 1 | 3 | 2 | *4 | *4 | | |
| H. pertussis | 0 | 2 | 2 | 1 | 5 | 3 | *4 | 0 | | |
| Bacteroides | 0 | 2 | 1 | | | 3 | | | | |
| Acid-fast | | | | | | | | | | |
| M. tuberculosis | 0 | 1 | 5 | 5 | 4 | 5 | 0 | | 5 | Combined strepto. & paramino salicylic acid |
| Spirochaetes | | | | | | | | | | |
| Treponema pallidum | 1 | 0 | 2 | 3 | 5 | 2 | | *4 | | |
| Borrelia | 1 | 5 | 5 | 3 | 3 | 3 | 0 | | | |
| Leptospira | 1 | 3 | 2 | 3 | 5 | | | | | |
| Rickettsiae | | | | | | | | | | |
| R. rickettsii | 0 | 0 | 1 | 1 | | 3 | | | | |
| R. tsutsugamushi | 0 | 0 | 1 | 1 | | 1 | | | | |
| R. prowazekii | 0 | 0 | 1 | 1 | | 1 | | | | |
| R. Akari | 0 | 0 | 1 | 1 | 3 | 2 | *4 | *4 | | |
| R. Burneti | 0 | 0 | 1 | 1 | | 3 | | *4 | | |
| R. Mooseri | 0 | 0 | 1 | 1 | | 1 | | | | |
| Large Viruses | | | | | | | | | | |
| Poxtacosis | 2 | | 1 | 1 | | | | | | |
| Lymphogranuloma venereum | 2 | 0 | 2 | 1 | | 3 | | | | |
| Prim. atypical pneumonia | 0 | 0 | 1 | 1 | | 1 | | | | |

1 = Sensitive (antibiotic of choice)

tomyacin with other agents is being tried as a possible means to avoid the development of resistant forms of tubercle bacilli and para-amino salicylic acid seems the most promising of these adjuvants at present.

Chloramphenicol (Chloromycetin) aureomycin and terramycin are best discussed as a group as they show many common characteristics. All three have a broad antibacterial spectrum demonstrated by their activity against both gram-positive and gram-negative organisms and their activity against certain rickettsiae and large viruses. All three are rapidly absorbed when taken by mouth and excreted in large quantities in the urine. All three are free from serious toxic properties, and development of resistance is not a frequent or serious problem as a rule. Although all three are derived from species of streptomycetes they are chemically different and differences in activity can be shown both in vivo and in vitro. Chloramphenicol is the drug of choice in the treatment of typhoid fever, aureomycin is preferred in the treatment of penicillin-resistant staphylococcal infections and terramycin is probably superior for the treatment of certain urinary tract infections. Apart from established advantages, the choice in any given infection will depend upon the sensitivity of the particular organism or organisms and upon possible sensitivities of the patient to one or the other of the antibiotics.

Tyrothricin (combination of gramicidin and tyrocidin) is too toxic for systemic use but has certain advantages for topical application. It is effective mainly against gram-positive organisms.

Bacitracin has a spectrum similar to penicillin and has the advantage of not being inhibited by penicillinase, which may be an important point in the treatment of mixed infections due to gram-negative and gram-positive flora, it seems devoid of sensitizing properties. Its main value is in local application (surgical wound infections, furuncles, carbuncles, abscesses, and pyogenic dermatoses). On parenteral injection it often

causes irritation and necrosis at the site of the inoculation, and nephrotoxic reactions seem to be high.

Polymyxins.—The practical experience with this group of antibiotics is so far very limited. They are reported to be active chiefly against gram-negative rods, and sensitive organisms do not appear to develop resistance, an advantage over streptomycin. Their chief use is hoped to be against pseudomonas infections which have been rather refractory to any of the above-mentioned antibiotics. The available preparations are toxic systemically but may be used topically.

This brief discussion of certain features of the antibiotics should convey to the student the importance of quick and accurate bacteriological diagnosis for any attempt at rational and successful chemotherapy. To achieve optimum conditions for bacteriological diagnosis the cooperation of the physician and surgeon with the laboratory is imperative. Proper collection of specimens is a fundamental step in providing suitable samples of sufficient quantity to carry out necessary examinations. Information on the clinical state and on any antibiotics already used is a prerequisite for the laboratory in the proper examination and reporting on specimens. Such considerations will be of great mutual assistance and prevent delays and disappointments for all involved, the patient, physician, and the laboratory.

In the following paragraphs the important pathogenic microorganisms in their relation to disease will be discussed. For the sake of uniformity the classification and terminology as given in Bergey's Manual (1948) will be followed, but long-established names in clinical use are given in brackets to avoid confusion, especially for those familiar with an accustomed terminology.

MICROCOCCUS PYOGENES (STAPHYLOCOCCUS PYOGENES)

The term *Staphylococcus* for the species of medical interest has been in use for more than 60 years and will probably persist

Staphylococci are parasitic on the skin and mucous membranes and about 20 per cent of people in some regions are nasal carriers. They are gram-positive spherical cells and occur characteristically in clusters. Staphylococcus grows on all ordinary culture media. A useful provisional differentiation of the pathogenic from saprophytic strains is obtained by means of the "coagulase test." Pathogenic strains coagulate human or rabbit plasma (coagulase positive). Because of their wide distribution and their relative resistance to drying and heat, infections with staphylococcus are very frequent and will be encountered by the surgeons in one form or another almost daily. A variety of clinical pictures are produced, ranging from mild superficial localized skin lesions to fulminating generalized infections. The ability of *Staphylococcus pyogenes* to cause disease is dependent on the toxins and enzymes produced by this organism, and the frequency and severity of the infections, as in most other diseases, depend on the relative susceptibility of the host and the defense mechanism of the body. A soluble, filtrable thermolabile exotoxin is produced by *Staphylococcus pyogenes*. This exotoxin is antigenic and gives rise to an antitoxin. By treatment with formalin, an antigenic toxoid can be prepared and can be used to produce active antitoxic immunity. The exotoxin of *Staphylococcus pyogenes* strains pathogenic for man contains a hemolysin for rabbit cells (alpha hemolysin), produces tissue necrosis, and is lethal for experimental animals.

Whether the exotoxin is a single entity or whether these three reactions are caused by different fractions of the toxin is a question which still awaits an answer. Leucocidin, another toxic fraction of staphylococcus pyogenes is of great importance for the establishment of infection because of its destructive action on leukocytes. This and coagulase are certainly partly responsible for the ease with which staphylococcus implantation occurs. Of the enzymes elaborated by *Staphylococcus pyogenes*, mention has al-

ready been made of the coagulase which permits the differentiation of pathogenic from nonpathogenic strains. Fibrinolysin is another enzyme produced by staphylococcus and seems to be confined to strains pathogenic for man. A mucolytic enzyme, hyaluronidase, is produced which increases the permeability of connective tissue and assists the spread of the infection from the initial focus.

Staphylococcus pyogenes is a good example of an organism which causes disease by active multiplication and toxic products. The clinical manifestations represent the sum total of both factors and the clinical features of severe infections somewhat depend on which predominates, toxemia or bacteremia with pyemic abscesses. If the defense mechanism of the host plays its proper part after the initial invasion, the infection remains localized. Staphylococcus infections occur in all age groups, but certain types are more common in children, e.g., osteomyelitis. *Staphylococcus pyogenes* is probably the most common cause of wound infections, either alone or in combination with other pathogens. Primary staphylococcus infections range from the most superficial folliculitis through furuncles, carbuncles and destructive osteomyelitis to fulminating septicopyemia. Many show a tendency to chronicity or persistence, particularly if the resistance mechanism is weakened by chronic diseases. Generalized spread may follow, either because of insufficient resistance or because the particular strain involved shows a high degree of invasiveness or toxin production. Thrombophlebitis and metastatic abscesses in practically any organ are a common sequence of generalized infection and occasion the severity of the clinical picture. The mortality rate of *Staphylococcus pyogenes* septicemia in the pre-penicillin days was as high as 50 to 90%.

Staphylococcal infections are characterized by the formation of abundant pus. The pus is of a creamy consistency, slightly yellow in color and consists mainly of poly-

morphonuclear leukocytes. Careful microscopic studies of stained films of the pus are often of help in assessing the state of resistance. Large numbers of engulfed cocci are a sign of good phagocytic response, whereas, destroyed leukocytes with masses of cocci outside should be a warning that the infection may spread if effective measures are not taken. Any accessible abscess should be drained before the bacteria spread either through the lymphatics or the blood vessels and cause systemic infection.

Penicillin is the drug of choice in acute staphylococcal infections if the strain is sensitive. Aureomycin follows as close second and takes first place if the strain is resistant to penicillin or in mixed infections. The dosage and mode of application depend on the type of infection, higher doses are usually necessary for the treatment of staphylococcal infections than for streptococcal or pneumococcal infections and in septicemia a dose of 300,000 to 600,000 units every 12 or 24 hours of procaine penicillin is required. Sound surgical judgment has to decide the indication for operation and adequate drainage. Acute localized infections in well vascularized tissues will respond readily to systemic treatment with penicillin. Infections of the bone and joints, and older abscesses with fibrotic avascular surrounding tissue, require local as well as systemic application after proper drainage in most instances. Staphylococcal empyema, brain abscesses and meningitis have been treated successfully with systemic and topical use of penicillin after drainage. Recently aureomycin has been used effectively in the treatment of staphylococcal brain abscess and septicemia. Bacitracin has been found effective in the treatment of staphylococcal abscesses. Meleney recommends the evacuation of pus and the instillation of Bacitracin 1,000 units/c.c.

In severe fulminating generalized infections with toxemia dominating the clinical picture, the use of antitoxin is indicated in combination with penicillin and/or aureo-

mycin. Antitoxin should be administered intravenously in such cases as early as possible.

In chronic and recurrent infections as well as in early stages of boils, staphylococcus toxoid has proved its value. The immunity produced by a course of toxoid is antitoxic and protects the tissues against the various destructive actions of the toxin. No general rules can be given as to how many courses may be necessary. Individuals vary considerably in their antitoxin response and adequate titer has to be judged in every case by clinical improvement. With care and patience most gratifying results can be obtained in recurrent furunculosis and chronic osteomyelitis.

STREPTOCOCCUS

The genus, *Streptococcus*, is of great importance in surgical practice and causes a wide variety of infections. Streptococci are gram-positive cocci which usually occur in chains of varying length and at times in pairs. A variety of species has long been recognized and many attempts at classification have been made. Differentiation according to changes produced on red blood cells has led to the terms hemolytic, viridans, and indifferent, which correspond respectively to *beta*, *alpha*, and *gamma* hemolysis. This caused overemphasis of hemolysis and the use of "hemolyticus" as a species epithet for which there is no justification. Actually there are five recognized species in the pyogenes group and two in the enterococcus group which are all hemolytic. Other classifications are based on the action of streptococci on a number of carbohydrates and other biological reactions, and on these Sherman (1937) divided the aerobic streptococci into four sections: (1) the hemolytic pyogenic group, (2) the viridans group, (3) the lactic group, (4) the enterococcus group. A subdivision of the hemolytic streptococci on the basis of antigenic differences has been developed by Lancefield (1928) and some of these subgroups differentiate a single species

while others comprise two or more distinct species. Lancefield subgroup D comprises all the enterococci whether they are hemolytic or not. No completely satisfactory classification has as yet been found for the viridans group and the anaerobic species. The growth requirements of streptococci vary for the various species, but blood agar usually provides a reasonably satisfactory medium for the isolation and characteristic colony production.

The variety of clinical signs and symptoms produced by streptococcal infections has led to the intensive search for the factors responsible for the differences both in the bacterial cell and in the host. Streptococci are widely distributed among man, animals, and probably plants. Some species are harbored as commensals in the human mouth and throat. Most species may survive at least for several weeks in clothing, bedding, and food. Milk is a favorable medium, and milk-borne streptococcal epidemics are not uncommon. Crowding as in schools, barracks, hospital wards, etc., increases the incidence of streptococcal infections. The two dread diseases of the surgeon and obstetrician, erysipelas and puerperal fever, are fortunately more of historical interest today, but the efforts of men like Holmes, Semmelweis, Billoth, Lister and many others should not be forgotten.

Extensive studies have led to the recognition of a number of components in the streptococcal cell which have contributed to our understanding of the disease processes. The serological classification of many aerobic streptococci depends on differences of a carbohydrate "C" substance, and although it has no relationship to virulence, it provides the grouping of streptococci as already pointed out. The vast majority of human infections seem to be caused by Group A streptococci although other groups occasionally cause disease. The groups can be further divided into types, and within Group A, for instance, at least 40 specific types have been identified. The type specific anti-

gen is a protein "M" substance, and antibodies against it confer type specific protection. The amount of "M" substance produced by a given strain is partly responsible for virulence. Type specific antigens in other groups are either proteins or polysaccharides. Two other fractions occur in the cells, T and P substances, which are not known to have any bearing on the virulence or pathogenicity of the strain. A number of other substances elaborated by streptococci have to be discussed briefly because they are essential for the understanding of the pathological features of streptococcal infections. The erythrogenic toxin, produced by streptococci, particularly of Group A, some of Groups C and G, is held responsible for the rash in scarlet fever. Immunity after scarlet fever is antitoxic, and only the erythrogenic toxin is neutralized, but no protective effect against the bacterial cell is provided. At least five immunologically distinct toxins are known which may explain the occurrence of an occasional second or even third attack of scarlet fever.

Most group A streptococci produce two different kinds of hemolysins O and S and the latter is pathogenic for laboratory animals. Antibodies against streptolysin 'O' can be demonstrated following infections with streptococci producing streptolysin 'O' and may persist for years. The part this 'O' lysin plays in the pathogenicity of the strain is not known, but streptolysin 'O' antibodies have been investigated for epidemiological studies.

Two enzymes, or pro-enzymes, produced by many strains of streptococci of Groups A, C and G, namely, streptokinase (fibrinolytin) and proteinase are both antigenic, but their relation to the lesions produced in infection is still obscure. Some strains elaborate hyaluronidase, an enzyme which is capable of breaking down hyaluronic acid, which forms the intercellular ground substance of connective tissue and cartilage. This enzyme was thought to be responsible for the invasiveness of streptococci, but

many highly pathogenic strains do not produce it, furthermore, many highly virulent streptococci are encapsulated and the capsule is formed mainly by hyaluronic acid.

Summarizing, we can only state at present that pathogenicity is associated with the "M" substance and the erythrogenic toxin. How much the other discussed components contribute to the virulence of a given strain remains to be proved.

Hardly any other bacterial species is capable of causing so many diverse pathological and clinical pictures as streptococci. Streptococci commonly enter through accidental wounds which may be quite small and hardly more than scratches. The response to this invasion depends on the strain and the susceptibility of the host. We have to differentiate between infections caused by the various groups of aerobic streptococci and the micro-aerophilic and the anaerobic streptococci.

The following clinical manifestations are caused in the vast majority of cases by *Streptococcus pyogenes* Lancefield Group A, more rarely by Group C, and very occasionally by Group G.

1 *Erysipelas*: The clinical picture is characterized by a *brawny-red* induration of the skin. The margin of the erythema is raised and irregular. Occasionally small blisters are formed on the surface, from which *Streptococcus pyogenes* can be isolated. If unchecked by treatment and in a patient without resistance, erysipelas can spread rapidly. Under such conditions, signs and symptoms of toxemia develop. Histologically the main feature of the lesion is intense inflammation of the superficial lymphatics which are filled with polymorphonuclear leukocytes, fibrin, and bacteria.

2. *Lymphangitis, lymphadenitis, cellulitis, and septicemia*: from a small puncture wound a red streak along the lymph vessel rapidly develops, the regional lymph nodes become tender and enlarged, softening and suppuration may follow. If spread is not checked bacteremia and septicemia may fol-

low, usually originating from a thrombophlebitis or directly from the lymphatics. Chills and high fever are the clinical signs of blood stream invasion, and other signs and symptoms may develop depending on the site or sites where infected emboli may be lodged. These may give rise to abscesses in the spleen, kidney, brain or to purulent arthritis, pleuritis, pericarditis, peritonitis or meningitis. This sequence of events may occur irrespective of the primary portal of entry. The upper respiratory tract is a frequent primary site of invasion and of major interest to the otolaryngologist. Pharyngitis, follicular tonsillitis, peritonsillar abscesses (quinzy), retropharyngeal abscesses, and the now rare condition of Ludwig's angina, are all caused by *Streptococcus pyogenes*. From the nasopharynx the streptococci may pass through the Eustachian tube to the middle ear and mastoid and to the large cerebral sinuses. Any of these may in turn lead to generalized infection. The involvement of the regional lymph nodes is very characteristic of streptococcal infection.

Recognition of the cause and proper counteraction have almost eliminated puerperal fever, a once very prevalent type of streptococcal infection, and if infection postpartum does occur immediate treatment will in most instances prevent serious sequelae.

Infections with *Streptococcus pyogenes* have fortunately lost much of their serious implication for the patient. Surgical drainage, wound débridement, and chemotherapy, either in the form of sulfonamides or in more serious cases in combination with penicillin will, in most cases, prevent spread and induce recovery.

Dosage and mode of application have to be adjusted to the individual case and should follow the principles as outlined in the general discussion. For superficial wound infections or infections of burns where local treatment may be a more rational form, topical application of tyrothricin or Bacitracin may be preferable to penicillin because of lack of sensitization.

The *viridans* group of streptococci is of much less concern in surgical practice. They probably play a role in infections of the gall bladder, apical abscesses of teeth, and are the cause of a certain number of urinary tract infections. Pathogenicity of the lactic group for man has never been established. In the treatment of any infection due to a member of the *viridans* group of streptococci it may be kept in mind that these organisms are less sensitive to penicillin and a higher dosage is required. Subacute bacterial endocarditis is considered a medical problem, with the exception of those cases where the vegetations are situated in patent ductus arteriosus and ligature or division and suture of the ductus is indicated. Intensive antibiotic treatment before and after operation will aid this procedure.

The last group of the aerobic streptococci comprises four species, commonly called *enterococci* or *faecal streptococci*, and all four species belong serologically to Lancefield Group D. Enterococci, as the name implies, form part of the flora of the intestines, particularly the colon. Their main importance is in infection of the peritoneum, the urinary tract, and in subacute bacterial endocarditis. Either alone, or more often with the colon bacillus, they are found in appendiceal abscesses, localized or diffuse peritonitis, following inflammatory processes or injuries to the terminal ileum, appendix or colon. In the urinary tract they cause cystitis, pyelitis and pyelonephritis. Invasion of the blood stream is rare but does occur, particularly following surgery on the infected kidney. Although enterococci are susceptible to penicillin they require much higher doses than the pyogenes group and in many instances the response is disappointing. Sulfadiazine has been used successfully in the treatment of urinary tract infections. Recent reports by several workers seem to indicate the superiority of aureomycin in the treatment of enterococcal infections (Long, 1950).

Meleney (1935) described a peculiar spreading burrowing undermining ulceration

of the skin caused by a micro-aerophilic hemolytic streptococcus. Treatment with zinc peroxide has been highly effective if the method as outlined by Meleney is carefully followed. "Progressive postoperative bacterial synergistic gangrene" is due to a nonhemolytic micro-aerophilic streptococcus (*Streptococcus evolutus*) in synergism with a *Staphylococcus pyogenes* according to Meleney (1919).

The several species of *anaerobic streptococci* vary in their biological character. They can be isolated from the mouth, intestines, and vagina of apparently healthy persons, and relatively little is known about their antigenic structure, and their pathogenicity for laboratory animals seems to vary widely. Their presence is often suggested by a foul odor and gas formation. Anaerobic streptococci are a frequent source of postpartum infection, either of a localized nature, as endometritis, or giving rise to generalized infections in which they can be isolated from the blood stream. Anaerobic streptococci are also often found in lung abscesses, usually in combination with other organisms, more rarely in mastoiditis and brain abscesses. Traumatic wounds with crushed tissues favor the growth of this group. Abscesses adjacent to the intestinal tract (perirectal, pilonidal cysts) are sometimes caused by anaerobic species which can also be found in mixed cultures in appendiceal abscesses and peritonitis. The pus and discharge are often but not always foul smelling. Most anaerobic strains of streptococci are sensitive to penicillin, but as they often occur in mixed infections, combined treatment with penicillin and streptomycin or one of the sulfa drugs may be a more adequate form of therapy. Drainage of any accessible accumulation of pus and removal of necrotic or gangrenous tissue are essential, and, depending on the severity of the infection and the tissues or organs involved, local and systemic treatment with the chosen chemotherapeutic agents may be indicated.

DIPLOCOCCUS PNEUMONIAE (PNEUMOCOCCUS)

The pneumococcus is a gram-positive, ovoid coccus, arranged in pairs and short chains and has an easily demonstrable capsule. Pneumococci can be grown fairly readily and on blood agar the colonies in aerobic culture produce a zone of greening. Bile or bile salts bring about rapid autolysis of the pneumococci, and turbid suspensions are cleared in a short time on incubation at 37° C. This simple test can be used to differentiate pneumococci from the viridans group of streptococci, the only organisms with which they could be confused. Pneumococci occur in the upper respiratory tract and cause infections mainly of the lungs, the accessory sinuses, middle ear, and meninges. Rarer but of greater importance to the general surgeon are pneumococcal peritonitis, metastatic purulent arthritis and empyema. As mentioned above, a characteristic of the pneumococcus is the capsule, and on the basis of differences in immunochemical structure of the capsular polysaccharide, pneumococci can be divided into at least 75 types. These capsular polysaccharides are antigenic, and type specific antibodies are protective. The capsule is an essential attribute for the pathogenicity or virulence of pneumococci by increasing the resistance of the bacterial cell to phagocytosis (Avery, 1932) (Wood, et al., 1946). No toxins in the accepted sense have been demonstrated. By means of specific antisera it is possible to determine the type of pneumococcus in a matter of minutes directly from the sputum or pus, and type specific antisera were the only effective treatment before chemotherapy. Even with the available chemotherapeutic agents antiserum occasionally remains a valuable adjunct. The physical appearance of pneumococcal pus differs from other pus by its high content of fibrin and its extremely tenacious character which is of practical importance because it hinders proper drainage and penetration of therapeutic

agents. This factor has led to interesting attempts to dissolve pneumococcal pus by the instillation of streptococcal fibrinolysin in order to facilitate access of sulfa drugs or antibiotics to the organisms.

The sulfa drugs and antibiotics have considerably lowered the mortality rate of pneumococcal infections. This is of great importance to the surgeon, as postoperative pneumonia was one of the serious consequences of many surgical interventions. Empyema, too, has become a relatively rare disease, and if it occurs can usually be well managed. The pus should be aspirated and replaced by local instillation of an antibiotic (penicillin) supplemented by systemic treatment if necessary. The same approach is successful in the management of pneumococcal arthritis. Aureomycin, chloramphenicol, and terramycin have also given good results in the treatment of pneumonia.

Pneumococcal meningitis used to be fatal in a very high percentage of cases. Recent reports by Dowling and his associates (1949) indicate that high doses of penicillin (1,000,000 units every two hours intramuscularly) may result in a considerably lowered death rate, 38% in their series as compared to 62% in a previous series in which smaller doses of penicillin were used systemically plus intrathecal injections. Other workers favor intrathecal as well as systemic treatment with penicillin. Oral aureomycin has been used successfully in a few cases of pneumococcal meningitis, but the numbers of cases are too few to date and no final conclusions can be drawn. Sulfadiazine, which penetrates freely into the spinal fluid, should always be used in addition to antibiotics. Pneumococcal meningitis remains a serious problem; primary foci, usually in the sinuses or mastoid, must be eliminated. Development of resistance of pneumococci to penicillin is a rare occurrence, but the possibility must be kept in mind, and if clinical improvement does not follow an ordinarily sufficient dosage of penicillin, aureomycin should be administered. As pneumococci

are carried by many people in the nasopharynx and cause a large number of sinus infections, the surgeon must be alert to the danger of pneumococcal meningitis in all cases of skull fractures.

ENTEROBACTERIACEAE

This family contains a number of species some of which live in the intestinal tract as commensals, others are obligatory parasites. All species are gram-negative rods and many show active motility by means of flagellae. All members of the family can be grown on ordinary culture media and ferment a variety of carbohydrates with the production of acid or acid and gas which, with other metabolic characters, are used to differentiate the various species. The lactose-fermenting genera are *Escherichia*, *Aerobacter* and *Klebsiella*, and the first two are often referred to as the Colon-aerogenes group. *Escherichia* can be divided into three species, *E. coli*, *E. freundii* and *E. intermedium*, but for the sake of simplicity we shall use the collective term *E. coli* for the three species. *E. coli* is an inhabitant of the intestinal tract of man and animals and as such is not only harmless, but, as already mentioned, is also useful in synthesizing certain vitamins (Johannson and Sarles, 1949). This point has certain practical implications for the surgeon. The preparation of the intestinal tract with sulfonamides and antibiotics before major surgical procedures on the colon has been adopted in many surgical departments and the decrease in bacterial flora, if protracted, may lead to vitamin K deficiency which in turn may elevate significantly the prothrombin time of the blood and cause hemorrhagic complications. The infections caused by *E. coli* are usually of a localized nature involving organs anatomically related to the intestines. *E. coli* is the most common cause of cystitis, pyelitis, and pyelonephritis. Of greater interest to the general surgeon are the infections in the abdominal cavity, peritonitis, and cholecystitis. In appendiceal abscess, *E. coli* can be

found in combination with a variety of other organisms; it certainly is not the primary cause of appendicitis. In peritonitis following perforation of some part of the intestine, *E. coli* is often the only or the predominating organism. In infants invasion of the blood stream followed by metastatic abscesses, particularly in the brain, is not a rarity. *E. coli* also plays a part, often in combination with other organisms (staphylococcus and streptococcus) in burns or in postoperative infections of abdominal wounds. The purulent discharge from *E. coli* infections often has a foul odor.

Removal of the primary focus of infection (appendix, gall bladder) and effective drainage of abscesses are as always the important steps. In the choice of an antibiotic, consideration must be given to the following points: *E. coli* is not sensitive to penicillin; moreover, many strains produce an enzyme, penicillinase, which inhibits this antibiotic. Therefore, either sulfa drugs or streptomycin have been used. Since aureomycin, Chloromycetin, and terramycin have become available, they may be considered the drugs of choice; they can be given orally and there is less danger of development of resistant organisms. They have the additional advantage of coping with mixed infections of many gram-positive and gram-negative organisms. If the sensitivity of the infecting pathogens can be determined quickly, the most effective drug can be selected. In the treatment of urinary tract infections, attention should be paid to any anatomical malformations or other occlusions which prevent free drainage and penetration, otherwise even the most effective antibiotic will only produce temporary relief.

The *Aerobacter* of different species produce, on the whole, the same type of infections as *E. coli*.

Klebsiella pneumoniae is a gram-negative, nonmotile rod. It grows well on ordinary nutrient media with large mucoid colonies. A striking morphological character is the capsule containing a type specific polysac-

charide, and Julianelle (1926) described three antigenic Types A, B, and C, and a heterogeneous Group X. Types A and B are much more frequently found than type C and seem to be more highly pathogenic. Friedlander's bacillus appears to live in the upper respiratory tract of man and has also been isolated from feces in healthy persons. Surgical infections caused by Friedländer's bacillus are empyema, bronchiectasis, sinusitis, occasionally appendiceal abscess, cystitis, and pyelonephritis. Brain abscesses and generalized septicemia with metastatic abscesses are rare complications. The exudates in Friedlander infections, unless frankly purulent, are highly viscous, due to the large amount of capsular material. Sulfadiazine and streptomycin have sometimes shown good results in the treatment of *Klebsiella* infections. Reports on the efficacy of aureomycin, Chloromycetin, terramycin and polymyxins in these infections are as yet too few to warrant definite conclusions, although good results have been obtained in a number of cases.

Salmonella typhi (typhoid bacillus) is a gram-negative motile bacillus, the causative agent of typhoid fever. It concerns the surgeon both during the active disease and in connection with lesions occurring years after the initial infection. The typhoid bacillus grows readily. It can be isolated from the blood of the patient during the acute stage (first week and beginning of second week) and from the urine and stool from the end of the first week on. Antibodies against various fractions of the bacillus appear in the blood, usually in the third week and may persist for several years after recovery, conferring a certain degree of immunity. The Widal reaction is based on the demonstration of these antibodies by agglutination of bacterial suspensions. The widespread artificial immunization against typhoid fever has somewhat impaired the diagnostic significance of the Widal test and the evaluation of positive results has to be based on at least 2 suc-

cessive tests at a few days' interval. Endotoxins which are liberated by autolysis of the bacilli are highly toxic and considered responsible for the clinical signs of chills, fever, malaise and headache.

The surgical lesions during the active stage of typhoid fever are orchitis, epididymitis, perichondritis and perforation of the intestinal ulcers with local or generalized peritonitis. Meningitis and empyema may also occur during the acute stage. It must be kept in mind that typhoid fever is a systemic disease and bacteremia is the main feature. Localization of organisms in practically any organ may result although the gall bladder, kidneys and bones are most commonly affected.

Chronic cholecystitis with or without acute exacerbations, often with formation of stones, is frequently due to the typhoid bacillus. Suppurative periostitis of the tibia, ribs or vertebrae may be a late sequel. The diagnosis can be established only by the isolation of *Salmonella typhi*. A few other members of the large *Salmonella* group may give rise to a similar clinical picture.

Chloromycetin is the treatment of choice for typhoid fever. The results in a large series of cases have been very gratifying and it is hoped that this treatment may prevent the carrier state. Only one other group of lactose-negative organisms of the Enterobacteriaceae family is of interest to the surgeon: *Proteus*. The members of this group are gram-negative motile bacilli which are widely distributed in nature, sewage and manure, and are frequently found in small numbers in the feces. Members of the proteus group are frequently the cause of urinary tract infections which can be extremely severe and lead to invasion of the blood stream, particularly following renal surgery. As one of the intestinal inhabitants, it is sometimes found in combination with other organisms in any infection of the abdominal cavity. The chief importance of the proteus group is as secondary invaders in

wounds, middle ear diseases, ulcerations and burns, particularly of the lower extremities (fecal contamination). Some members of the proteus group are highly proteolytic, and any attempt at skin grafting while the area is infected by these microbes is futile. Wound exudates of proteus infections have a characteristic unpleasant odor. These infections, although as a rule not serious on the skin, are very stubborn. Some strains are sensitive to streptomycin, some to Chloromycetin or terramycin and treatment with either may be effective, but sensitivity varies much from strain to strain. The local application of lactic acid 5 to 10% is a useful form of treatment if the strains are not susceptible to available antibiotics. Aurcomycin has been without value in the cases reported.

Although not belonging to the family of Enterobacteriaceae but to the Pseudomonadaceae, the only member of clinical importance, namely, *Pseudomonas aeruginosa* (Pyocyaneus), is discussed in connection with proteus, because they quite often occur together in mixed infections and have other features in common. *Pseudomonas aeruginosa* is a gram-negative, usually motile bacillus, which produces a highly diffusible greenish or greenish-blue pigment, and this organism has been long known to the surgeon as "blue pus" former. Like proteus, it is widely distributed in nature and occurs occasionally in the intestine. Similar to proteus it causes infections of the urinary tract, the middle ear, and particularly infections of burns and wounds. Meningitis, arthritis and eye infections have been reported. The bluish green color and a sweetish smell of the exudate is very characteristic. As far as chemotherapy is concerned the same rules apply as for proteus infections, but polymyxins appear to be more uniformly effective against this microbe. For local treatment citric instead of lactic acid can be used. In prolonged chronic infections with either of these organisms an autogenous vaccine should be considered; the response is in many cases very gratifying.

NEISSERIA GONORRHOEAE (GONOCOCCUS)

The gonococcus appears in exudates and cultures as a gram-negative diplococcus with the adjacent sides flattened (coffee-bean shaped). The cultivation of the gonococcus presents certain difficulties, as it is fastidious and easily killed by drying or sunlight. Great care has to be taken in securing exudates, and best results with culture methods are obtained if the appropriate media are inoculated at the bedside. In acute disease of the male the finding of large numbers of morphologically typical intracellular or extracellular organisms on stained films may seem sufficient corroboration of the clinical diagnosis. The surgeon is as a rule confronted with complications or sequelae of the primary gonococcal infection. Infection of the urogenital tract in the female is a frequent complication of gonorrheal cervicitis; urethritis, infection of Skene's or Bartholin's glands, salpingitis, and peritonitis are the lesions caused by lymphatic spread or direct progression. A creamy mucopurulent discharge is characteristic of gonorrheal infection. Fibrotic occlusion of the Fallopian tubes may necessitate surgical measures. In the male, prostatitis, urethral strictures and epididymitis are the common complications of urethritis. Invasion of the blood stream by the gonococcus may take place and produce arthritis, fibrositis, iritis, endocarditis, pericarditis, and occasionally meningitis. In the acute state, penicillin, 200,000 to 300,000 units, will cure the majority of cases. Localized complications, such as arthritis or Bartholin abscesses may require surgical drainage and local instillation of penicillin as well as systemic treatment.

PARVOBACTERIACEAE

The family consists of several genera and includes a large number of species, all of which are primarily of interest to the physi-

cian A few species cause abscesses and other lesions which require surgical attention.

Pasteurella.—*Pasteurella multocida* and *Pasteurella tularensis* appear to be the two species which need to be considered. Both are gram-negative, short, nonmotile, oval bacilli; the latter may show great pleomorphism. *P. multocida* grows readily but *P. tularensis* needs special media for primary isolation and will be missed unless there is some indication given to the laboratory of the suspected type of infection. *P. multocida* occur as saprophytes and parasites in many animals and can be found frequently in the upper respiratory tract of normal domestic animals. Human infections in the form of abscesses, frequently complicated by osteomyelitis, are generally due to animal bites, particularly cat, dog and rabbit. Generalized infections, cases of meningitis and empyema have been reported. All the strains of *P. multocida* which have been recently tested were sensitive to penicillin (Schipper, 1947).

P. tularensis is the causative agent of tularemia. The main sources of infection for man are: (1) direct contact with infected animals, (2) the bites of ticks and flies (deer flies), (3) ingestion of contaminated meat or water, (4) laboratory infections. Some occupations show a higher incidence of infections: butchers, hunters, farmers and laboratory workers. If the organisms penetrate the skin, a primary lesion in the form of an ulcer develops in a certain percentage of cases. Spread occurs along the deep and superficial lymphatics leading to lymphangitis and lymphadenitis with suppuration. This is the type of tularemic infection which the surgeon encounters. Generalized signs of fever and elevated sedimentation rate are always present, the white count may or may not be elevated. A transitory bacteremia is usual during the first week but disappears as soon as antibodies are formed, but spread of the infection by the lymphatic route may take place with renewed, usually fatal, invasion of the blood stream, unless checked by treatment. The diagnosis can be made

by the isolation of *P. tularensis* from the primary lesions or the pus from lymph nodes in the early stages. As already stated *P. tularensis* requires special media, and for this reason and to safeguard the laboratory worker from infection, the suspicion of such an infection must be clearly indicated on any specimen sent to the laboratory. Recovery from tularemia confers a high degree of immunity. If isolation of the organism fails, the clinical diagnosis can be confirmed in the later stages of the disease by serological methods. Agglutinating and complement fixing antibodies appear in the blood from the second week on and show a rising titer during the third and up to the fifth week. These antibodies may persist for many years or even through life. A positive test, therefore, does not necessarily indicate present infection with *P. tularensis*, but the rise in titer between second and third week of a disease suspected to be tularemia is of a great diagnostic significance. An intradermal test with specially prepared antigen (Foshay, 1940) is of diagnostic value. It shows the tuberculin type of reaction 48 hours after inoculation and is usually positive earlier than the agglutination test. This altered skin reactivity may persist for the remainder of life. Tularemia had been a highly fatal disease until streptomycin became available, this antibiotic has an almost immediate effect and a short course of 6 days has been found effective, even in the pneumonic form of tularemia, if treatment is started early in the course of the disease. Aureomycin has been used in a number of cases with very satisfactory results (Woodward et al., 1949).

Brucellae.—*Brucella suis*, *Brucella abortus* and *Brucella melitensis* are the cause of acute and chronic brucellosis, a relatively common disease on the North American continent. Although, primarily a systemic disease with which the physician has to deal, brucella infection may lead to localized lesions in bones and joints and as such becomes the concern of the surgeon. Because of the great difficulties in diagnosis, it appears im-

portant to draw the attention of the surgeon to this type of infection and make him conscious of the fact that brucellosis gives rise to a variety of lesions.

Brucella is a gram-negative, small, non-motile rod, which causes disease in Ungulates transmissible to man. The difficulties of isolation, particularly in chronic cases, have been stressed by all workers, and *Brucella abortus* requires increased CO₂ tension for isolation. The main source of infection is unpasteurized milk, milk products, and infected meat. The localized lesions are mainly granulomatous in character, and proliferation of the cells of the reticulo-endothelial system forms the basis of the lesions. The clinical picture has so many variations that the multiplicity of symptoms as such is often suspicious. The only exact diagnosis can be made by the isolation and identification of the organism which, as already mentioned, is beset with difficulties. Skin reaction to *Brucella* vaccine or Brucellergin and the agglutination test and the Opsonophagocytic Index are diagnostic adjuvants, but only of relative value. As immunity reactions may persist for many years, a positive test is only evidence that exposure to *Brucella* occurred at some time but does not necessarily indicate an active infection. Negative results, on the other hand, do not exclude active brucellosis (Huddleson, 1943). Bone and joint complications of brucellosis are probably not very frequent, but the small number of reported cases may be misleading because of the rarity with which the diagnosis can be established with certainty. Arthralgia in multiple joints seems to be a common symptom, but suppurative arthritis, osteomyelitis, and spondylitis have only more recently been given proper attention. Every attempt should be made to verify the clinical diagnosis by the isolation of the microbe in order to institute therapy. Streptomycin in combination with sulfadiazine proved to be a fairly effective form of treatment, but is being replaced by aureomycin, Chloromycetin and terramycin. These antibiotics have

shown very good results in the treatment of acute brucellosis, although the evaluation of results in chronic cases is much more difficult and has to await further trial.

Haemophilae influenzae is a small gram-negative rod of variable length and requires media containing growth factors X and V for its isolation. Six types, A, B, C, D, E, F, have been identified by differences in capsular polysaccharide. No exotoxin has been found and the pathogenicity appears connected with the specific capsular substance, very much as in pneumococcal infections. Severe infections, particularly in infancy and childhood, are caused by type "B" in the majority of cases. Nontypeable strains are frequently found in the upper respiratory tract and accessory sinuses in children and adults. The role of these strains in acute and chronic infections of the lungs and upper respiratory tract is still a controversial question, although clinical disease is often improved after their elimination.

H. influenzae, type B, in children causes bacteremia and a severe form of meningitis and frequently arthritis; obstructive tracheitis, pneumonia, and empyema are less common.

The invasion of the blood stream takes place from the nasopharynx, but pharyngitis and otitis media are less severe complications. The diagnosis and type are established by the isolation of the organism and subsequent capsular swelling test with type specific rabbit antisera or direct capsular swelling in the exudate or spinal fluid. The mortality rate of *H. influenzae* meningitis has been considerably reduced (from about 90% to about 20%) by the combined use of sulfonamides, streptomycin, and rabbit antiserum (Alexander and Leidy, 1947), but it still remains a serious disease and frequently gives rise to brain abscesses. Aureomycin and sulfadiazine have been used effectively in some cases. Adequate drainage of abscesses, empyema and purulent exudate in arthritis is indicated and should be followed by local and systemic antibiotic treatment.

Bacteroidae.—*Bacteroides* are small anaerobic gram-negative pleomorphic non-spore-forming bacilli, their normal habitat is on the mucous membranes of the body. They cause putrid infections of wounds either alone or in combination with other bacteria. Postpartum infections, tonsillar abscesses, and subphrenic abscesses are the main lesions caused by this genus. Blood stream invasion from any of these foci may occur, with metastatic abscess formation and a high fatality rate in the reported cases. Anaerobiosis is essential for isolation, and it seems probable that such infections are missed unless anaerobic cultures are routine in the laboratory. McVay and co-workers (1949) reported two cases successfully treated with aureomycin. In vitro test showed the organisms (*B. funduliformis*) resistant to penicillin, sulfonamides, and streptomycin.

BACILLACEAE

Only one species of the aerobic genus *Bacillus* is pathogenic causing the infection known as anthrax. *Bacillus anthracis* is a facultative aerobic gram-positive spore-forming, capsulated, nonmotile bacillus, occurring often in long chains. Growth occurs readily on conventional laboratory media. The spores are very resistant and may survive for years in earth or other materials. Anthrax is primarily a disease of domestic animals; human cases are relatively rare and occur mainly in men occupied in certain industries dealing with hides or skins, or in agricultural workers. Shaving brushes and furs may be rare sources of infection. Cutaneous anthrax (malignant pustule) starts as a small red spot which shows a central blister and some edema. In the blister fluid the anthrax bacillus can be readily found. During this stage the nature of the lesion may not be recognized unless the occupation of the patient rouses suspicion. In later stages the necrotic center of the lesion with the black crust "eschar," and the usually marked edema are characteristic of anthrax. Enlargement and

tenderness of the regional lymph nodes are frequently present, and depending on the severity of the infection, systemic signs and symptoms of fever and prostration occur. The bacteriological diagnosis is made by direct culture or inoculation of exudate into mice or guinea pigs and subsequent culture. In severe unchecked infections bacteremia occurs, and the mortality rate of such untreated cases may be as high as 20 per cent. Surgical incision of the primary lesion is contraindicated, and early recognition of the true nature of this infection will prevent dangerous interference. Penicillin is the drug of choice, and treatment should be maintained at least until bacteria disappear from the lesion. Good results have been obtained with antiserum.

CLOSTRIDIUM

Clostridium is of much greater practical importance to the surgeon. This genus contains a large number of species of which only some are pathogenic for men, among them the organisms connected with gas gangrene and tetanus. The clostridia are gram-positive, anaerobic rod shaped organisms producing endospores; many species are motile. They occur naturally in soil and the intestinal tract of man and animals. Under anaerobic conditions most species grow on a variety of media and can be differentiated on the basis of biochemical reactions. The clostridial species most commonly present in gas gangrene elicit their effect in the tissues, and the systemic signs and symptoms, by toxins and enzymes produced by the bacterial cells. In most infections more than one pathogenic species is present and various nontoxin-producing saprophytic species occur in the same wounds (MacLennan, 1944). The following species are regarded as primary cause of gas-gangrene (Reed, 1949); *Cl. perfringens* (Welch's *Bacillus*), *Cl. novyi*, *Cl. septicum*, *Cl. bifermentens* (*Cl. sordellii*) and rarer *Cl. histolyticum*. Other organisms (gram-positive cocci and gram-negative rods) are frequently present in the wounds

with clostridia. As all clostridia require more or less strict anaerobic conditions, special features of the wounds are essential for their implantation and multiplication. Crushing injuries of the tissues, with foreign bodies, bone splinters, clothing, and soil particles provide an ideal basis, and the virulence of the particular species introduced determines the severity of the infection. Local defense mechanisms of the host seem to be interfered with by the necrosing and leukocidal toxins. The isolation of pathogenic clostridia from the wound does not necessarily imply clinical gas gangrene as there are wide variations in toxigenicity of different strains in the same species. The five mentioned species of *Clostridium* elaborate soluble exotoxins. *Cl. perfringens* has been divided into four types, A, B, C, and D on the basis of seven different toxins, but only type A has so far been isolated from human infections. Type A produces so-called alpha toxin which has been identified as lecithinase and forms the main lethal component of culture filtrates. Other enzymes produced by *Cl. perfringens* are hyaluronidase and kappa toxin, a collagen-splitting enzyme, and a variety of additional enzymes have been isolated and studied. All these enzymes appear to be of importance for the growth and multiplication of *Cl. perfringens* in the tissues and the majority of the histological changes in the muscles can be produced with culture filtrates. Other pathogenic species of clostridia have also been studied as to their toxin and enzyme systems (DeSpain Smith, 1949). All elaborate soluble toxins which are neutralized by specific antitoxins. The toxins have lethal, necrotizing and hemolytic actions either due to a single or to multiple substances.

The clinical picture of gas gangrene infection is characterized by edema of the subcutaneous tissues; the muscle fibers are friable and discolored, gas bubbles may appear between muscle fibers and the surrounding tissues and account for "crepitation." A blood-stained, often foul smelling, thin

exudate is discharged, containing gas bubbles. If highly proteolytic species take part in the infections, rapid and widespread tissue destruction takes place. Systemic signs and symptoms of severe toxemia with leukopenia, fever and high pulse rate may be marked. Invasion of the blood stream by one or more species can occur. The clinical picture is not characteristic for any specific type of clostridial infection, but the local lesion in infections by a single species is often characteristic and very different from that of mixed infections. In clinically established gas gangrene, time is of the utmost importance, and treatment with antitoxin must be started at once. Polyvalent antitoxin (*Cl. perfringens*, *Cl. septicum* and *Cl. novyi*) standardized in international units should be given either intravenously or intramuscularly, depending on the gravity of the infection. The antitoxins are prepared by immunization of horses, and the necessary precautions have to be taken to prevent serious incidents due to hypersensitivity. The minimal recommended dose in established infections is 3 times the prophylactic dose (9,000 units *Cl. perfringens*, 4,500 units *Cl. septicum* and 3,000 units *Cl. novyi*). This dose should be repeated every 4 to 6 hours according to the response of the patient. Chemotherapy, sulfonamides and penicillin must be considered an adjuvant only to surgery and antitoxin. Local applications of sulfadiazine and penicillin as well as systemic treatment is advisable, because relatively little will penetrate into the ischemic parts from the circulation.

Clostridium tetani belongs to the same genus as the organisms causing gas gangrene, but provokes an entirely different clinical picture. *Cl. tetani* is a slender gram-positive bacillus with terminal spores and requires very strict anaerobic conditions for growth. It is widely distributed in soil and occurs occasionally in the feces of man and horses. Due to the almost universal use of toxoid for active immunization, tetanus has become a very rare disease, and many a surgeon may not encounter a case during his active life,

but on the other hand the very fact of its rarity may cause diagnostic difficulties in the critical early stages of tetanus with possible fatal consequences for the patient. Tetanus is a typical example of a pure toxemia, all clinical signs and symptoms are due to the action of the toxin and not to the multiplication of the bacteria. Tetanus may follow certain penetrating types of wounds, infections of burns and severe frostbites, or, fortunately, rarely surgical procedures, particularly in abdominal surgery and extractions of teeth. An interesting case has been reported by Murray and Denton (1948) in which clinical tetanus was almost certainly contracted from plaster of Paris. Certain conditions of the wound favor, and are almost a prerequisite for, the germination of *Cl. tetani*. Deep penetrating wounds with associated infection or foreign bodies provide the anaerobic environment which is necessary but the wound may be very small and even apparently healed by the time clinical tetanus develops. It has been shown experimentally that if washed spores are introduced into healthy tissues they are taken up by phagocytes and may be eliminated, but if the tissues are damaged and necrotic, the spores germinate and toxin is elaborated. It should be remembered that spores may persist in the tissues for a considerable time. If and when the toxin reaches the anterior horn cells of the nervous system, clinical symptoms of tetanus become manifest. The pathway of the toxin is still a controversial subject; two main hypotheses exist. (1) toxin is absorbed by the motor nerve endings and reaches the anterior horn cells through the axis cylinders; (2) toxin is carried by the arterial blood to the central nervous system. The latter theory is receiving most support at present. The incubation period in tetanus varies considerably and may be as short as 3 or 4 days or as long as several weeks with an average of 10 days. The prophylactic use of tetanus antitoxin after injuries will either prevent tetanus completely or pro-

long the incubation period, and the clinical disease will be milder.

Clinically, stiffness of the neck, trismus, and difficulties in swallowing are often the first symptoms. Tonic contractions of muscles and very painful clonic spasms elicited by very trivial external stimuli follow. Contractions of the facial muscles produce the characteristic "risus sardonicus." In later stages all the muscles are affected and death is due to respiratory failure. The temperature may be slightly elevated, the pulse is rapid, and the patient remains conscious until the end.

The clinical diagnosis should not present any difficulties even in the initial stages if the surgeon is alert to the possibility. The bacteriological proof is often very difficult or even impossible without adequate specimens. As previously mentioned the initial lesion may be completely healed, but even if a wound is present, relatively large amounts of tissues and debris from the depth of the wound must be available for cultures to provide some chance for the isolation of *Cl. tetani*.

Treatment.—Once clinical signs and symptoms of tetanus are pronounced, the chances of recovery are slim, but good results have been obtained with large doses of antitoxin by various routes, provided complete removal of the focus is achieved. Constant sedation (paraldehyde, chloral, etc.) and muscle relaxants (Curare) are essential to prevent exhaustion. Supplementary oxygen therapy is important. Antitoxin should be administered intrathecally (10-20,000 units) intravenously 20-50,000 units and 50-80,000 units intramuscularly in the first 24 hours. The intravenous and intramuscular doses must be repeated and carried on depending on the progress of the patient. Even more heroic doses will not save every patient, and the chief effort of the physician must be the prevention of tetanus by the prophylactic use of antitoxin (1,500 units) as soon as possible after injuries occur. Tetanus antitoxin is horse serum (actively im-

munized) and the sensitivity of the patient must be carefully checked, and if indicated, desensitization must be attempted, but in any case antitoxin cannot be withheld. Penicillin should be used to prevent secondary infections (pneumonia), and as *Cl. tetani* is sensitive to penicillin it may check multiplications in the focus, but it has no effect on the toxin absorption.

CORYNEBACTERIACEAE

Corynebacterium diphtheriae is a gram-positive, nonmotile nonspore-forming bacillus often club shaped and usually arranged in palisades. Growth can be obtained under aerobic conditions on the usual laboratory media, but Tellurite-containing media are generally employed to suppress the growth of other organisms and to permit differentiation of the three recognized types of *C. diphtheriae* (gravis, mitis and intermedius). It produces a powerful toxin which is responsible for the disease. Diphtheria is transmitted through direct contact by droplets either from the active case or a carrier.

The incidence of diphtheria has been reduced by widespread immunization with toxoid. The organisms multiply to some extent in the superficial layers of the mucous membranes and elaborate their toxin which has a highly necrotizing effect on the cells. The local tissue response to the toxin consists of a fibrinous exudate which forms very adherent greyish tough membranes. Tracheotomy has to be performed if obstruction of the air passages by rapidly spreading membranes occurs. For cultures, pieces of the membrane should be sent, but in the classical form of pharyngeal or nasal diphtheria, bacteriological diagnosis is mainly confirmatory, and treatment with antitoxin must be started on the basis of the clinical signs and symptoms, the unavoidable delay of the bacteriological method involves too great a risk for the patient and it is much safer to give antitoxin once too often than to withhold it even for a day where it is required. Occasionally

wounds may become infected with *C. diphtheriae*, and because of the rarity of such infections in temperate climates, they frequently remain undiagnosed until the appearance of late paralysis causes suspicion. Every wound with a greyish slough or membrane and no tendency of healing should be suspected and cultured to arrive at the correct diagnosis. Penicillin is used as an adjuvant with antitoxin to take care of any simultaneous streptococcal infections and in the hope of lowering the carrier incidence. It is most certainly not a substitute for antitoxin.

ERYSIPELOTHRIX

Erysipelothrix rhusiopathiae cause *Erysipeloid*, a usually harmless cutaneous infection. The causative organism is a gram-positive nonmotile, nonspore-forming bacillus. Growth is obtained under reduced oxygen tension on simple media or better on enriched media. Butchers and persons handling poultry and fish are prone to this infection. A purple erythema, with some edema and a slightly elevated margin, is characteristic of this lesion, which occurs chiefly on the hands. The infection remains localized as a rule although peripheral extension with central clearing is common. *E. rhusiopathiae* is sensitive to penicillin, and the use of it probably shortens the course, although the infection is self-limiting and treatment is not a necessity.

MYCOBACTERIACEAE

Mycobacterium tuberculosis of either human or bovine type is responsible for the clinical disease of tuberculosis. With the discovery of this bacillus in 1882 by Robert Koch a common cause was found for an extraordinarily wide variety of lesions and clinical symptoms. Surgeons and physicians are equally concerned in the diagnosis and treatment of tuberculosis. *M. tuberculosis* is a slender bacillus, nonmotile and nonencapsulated. Its most characteristic property is the resistance to decolorization by acids;

"acid fastness" Special media are used for the isolation of the tubercle bacillus from exudates or tissues, and growth is characteristically slow. Recently media have been developed (Dubos and Middlebrook, 1947) which permit more rapid growth.

There are two main routes by which the tubercle bacillus gains entrance into the body (1) by inhalation, (2) by ingestion. A third route is possible, namely, inoculation into the skin through small wounds; this last mode of infection is rare and causes usually only the verrucous type known as tuberculosis verrucosa cutis, found on the hands of pathologists and butchers, as professional hazards from infected cadavers. Tuberculosis illustrates well the importance of host-parasite relationship. The interaction between the bacterium and the human or animal body is the pertinent factor rather than any special constituents of the bacterial cell. In the various infections so far discussed it has been pointed out, how either toxins or other cell factors are responsible for the pathogenicity and disease manifestations. As yet there is no convincing evidence that a toxin or any other specific cell factor in the tubercle bacillus is responsible for its pathogenicity, although "toxic" substances may well be produced during its multiplication in the tissues. The protein fractions of the bacterial cell are of special interest because they are responsible for the "tuberculin" reaction. The injection of either Koch's old tuberculin (O.T.) or of the purified protein derivative (P.P.D.) (Seibert, 1934) elicits a characteristic delayed type of response in the individual who has been infected; it does not necessarily indicate active infection. A negative tuberculin test excludes active infection with two possible exceptions: an overwhelming generalized disease, or the very early stages of a first infection (3 to 4 weeks). Tuberculosis caused by the bovine or human type cannot be distinguished on the basis of the tuberculin test. The pathogenesis of tuberculosis is a very intricate subject and cannot be discussed

even superficially in this chapter. The reader should refer to general textbooks of pathology or to special monographs (Rich, 1944).

Tuberculous lesions are either productive or exudative in nature, but both types may be present at the same time in the same organ. Microscopically and histologically the productive type of lesion is represented by "tubercle" formation. The exudative type resembles in its very early stages the inflammatory reaction caused by pyogenic organisms, and exudation of fluid and polymorphonuclear cell infiltration are its chief characteristics. This latter type of cell is soon replaced by mononuclear forms. Tuberculosis of certain organs is primarily a concern of the surgeon. These include the lymph nodes, bones and joints, kidneys, epididymes, prostate and seminal vesicles, the pelvic organs in the female, the intestinal tract, the serous membranes (peritonitis, pleuritis, pericarditis), the central nervous system (meningitis and tuberculoma), the larynx and trachea. The clinical features, the differential diagnosis and the surgical procedures are dealt with in the special chapters. The diagnosis rests ultimately on the demonstration of virulent tubercle bacilli in the exudates or tissues. The finding of acid-fast bacilli on smears is presumptive but not conclusive evidence of tuberculous infection. Saprophytic mycobacteria and acid-fast strains of the genus *Nocardia* may resemble tubercle bacilli so closely that they cannot be differentiated on stained films. Relatively large numbers of organisms have to be present in a specimen to be detected on a smear, and cultivation and animal inoculation must be resorted to if early lesions are to be diagnosed. The early diagnosis of tuberculosis is of utmost importance not only for the sake of the patient but also for epidemiological reasons. The histological examination will often be of great help, but if tissue is being removed, part should be used for animal inoculation which will prove conclusively the presence or absence of virulent tubercle

bacilli. X-ray examinations and tuberculin tests have only a limited significance compared with the cultural methods and pathogenicity tests. However, even one or two negative cultures do not rule out tuberculosis.

With the discovery of streptomycin by Waksman and Schatz (1944) active treatment of tuberculosis has become a reality. No longer need the physician and surgeon trust only to the defense mechanism of the body to overcome the infection with the help of supportive means like rest, nourishment, etc. Streptomycin, Dihydrostreptomycin and probably Neomycin, if wisely used, can alter the prognosis of a large number of cases and prevent many crippling sequelae of this disease. In spite of certain limitations (toxicity and development of resistance) the therapeutic value of streptomycin in the treatment of tuberculosis is firmly established. Reviews on the chemotherapy of tuberculosis on thousands of cases have been published (Pfuetze and Pyle, 1949) (Committee on Chemotherapy and Antibiotics, 1949). The problem of toxicity has been somewhat reduced by the use of smaller doses over shorter periods of time and the combination of streptomycin with para-amino-salicylic acid seems to decrease the development of resistance. Neomycin (Waksman and Chevalier, 1949) is active against *Mycobacterium tuberculosis*, even against strains which are resistant to streptomycin, and development of resistance to it appears to be limited.

Streptomycin in itself is not a cure for tuberculosis, but should be considered a very valuable adjunct to other established forms of treatment. In milary tuberculosis and tuberculous meningitis it is the only promising treatment available, although the results have on the whole been disappointing. It is of great value to the surgeon as preparatory treatment before chest surgery and in the treatment of tuberculous sinuses and tuberculosis of the bones and joints. Indeed, most forms of tuberculosis are benefited by streptomycin improving the general condi-

tion of the patient, thus making surgery possible. Cavitation, extensive fibrosis or caseation are as such usually not improved, and streptomycin is not indicated in such lesions.

At present streptomycin is used in doses of 0.5 to 1 Gm. daily, divided into 2 doses for a period of 42 to 120 days, depending on the severity of the case. For the treatment of sinuses or in joint and bone lesions, local instillation as well as systemic treatment may be indicated. In tuberculous meningitis intrathecal treatment (50 to 100 mg.) every 24 hours to 48 hours, as well as systemic streptomycin is used by many (Cairns, et al., 1950). Para-amino-salicylic acid (15 to 20 Gm. daily) combined with streptomycin or following streptomycin is widely used. It must be realized that these treatment schemes are empirical only and the optimal method has yet to be determined.

ACTINOMYCES

Actinomyces bovis is the cause of actinomycosis in man. *Actinomyces bovis* is a gram-positive branching filamentous nonmotile organism. No spores are formed. Pus from actinomycotic lesions frequently contains "sulphur granules," whitish or yellowish firm granules which, when slightly crushed and viewed under the microscope, show entangled filaments with swollen clublike ends. This appearance, which is also characteristic in histological sections is responsible for the name "ray fungus." *Actinomyces bovis* can be grown on usual laboratory media under anaerobic conditions. Growth is slow, taking generally 4 to 6 days at 37° C. *Actinomyces bovis* seems to be a strict parasite and has not been found outside the human or animal body. It can be found in scrapings from the teeth, gums, and tonsillar crypts in apparently healthy persons, and the disease is due to an endogenous infection. Although the exact pathogenesis is little understood, trauma, e.g., tooth extractions, fractures of the jaw or other injuries to the mucous membranes of the mouth, appears to favor the in-

vasion of *A. bovis*. The relative rarity of the disease suggests that as yet unknown factors must play an important role.

Actinomycosis is a subacute or chronic, slowly progressive disease, with three main localizations the *cervico-facial*, *thoracic* and *abdominal* regions. The first form is the most common and affects the soft tissues of face and neck. The lesions show marked induration and a peculiar hard consistency is characteristic. The skin over the lesions is swollen and brownish red in color. If central softening and break through the skin occur, chronic fistulas are formed. The *abdominal type* starts usually in the appendiceal region and is commonly mistaken for a simple appendicitis. After appendectomy, the poor healing of the wound and fistula formation and later the formation of a tumor-like mass should arouse suspicion. *Thoracic actinomycosis* affects the lungs, forming abscesses and cavities. All three forms, if undiagnosed and untreated, will spread slowly but relentlessly, even eroding bone. Hematogenous spread with metastases in many organs has been observed. The clinical diagnosis of actinomycosis can be verified only by the cultivation and isolation of *Actinomyces bovis*. The microscopic demonstration of gram-positive filaments or of typical "ray fungus" in histological sections is acceptable as provisional diagnosis, but cultural isolation should be at least attempted. This presents usually no difficulty in material from primarily closed lesions, provided the laboratory receives some indication of the presumptive diagnosis from the surgeon and cultures are kept long enough. The isolation from heavily contaminated material is more difficult, but if proper methods are used it is generally successful. The interpretation of positive findings from sputum requires caution because as pointed out, the mouth is the normal habitat of *A. bovis*.

Penicillin is at present considered the treatment of choice. Rather high doses have been

recommended, 500,000 to 1,000,000 units a day (Nichols, 1948). Surgical excision or drainage may be indicated in some cases.

NOCARDIA

Some species of the genus *Nocardia* are pathogenic for men, causing chronic suppurative or granulomatous lesions. *Nocardiae* are gram-positive rods and branching filaments, some species are partially acid fast and these may be mistaken for tubercle bacilli in stained films. In pus or exudates from lesions granules similar to the "sulphur granules" in actinomycosis can be found. *Nocardia* can be grown on simple media under aerobic conditions but growth is slow.

Nocardia occur freely in nature, and infection is either airborne through inhalation or by introduction into the subcutaneous tissue through trauma. Pulmonary nocardiosis resembles tuberculosis both by x-ray and in clinical symptomatology. Hematogenous spread leads to metastatic lesions throughout the body. Brain lesions and meningitis may be caused by *Nocardia* either as primary lesions or as metastatic lesions from the lungs. In the majority of reported cases the diagnosis was made only after death. The lesions of the subcutaneous tissues and bones are easily mistaken for actinomycosis or fungus infections. Multiple draining sinuses are characteristic of these lesions, which result in the clinical picture of Madura foot. Generalized granulomatous disease due to a *Nocardia* species has been reported recently by Cuttino and McCabe (1949). The frequency of *Nocardia* infections is difficult to estimate, but it is probably higher than the reported cases would indicate. The pulmonary type has had a very high mortality rate. The correct diagnosis can be made only by the isolation and identification of the organism. If the diagnosis is made early enough, sulfadiazine seems to be beneficial (Kirby and McNaught, 1916). One species, *Nocardia asteroides*, has been found susceptible to chloramphenicol (McLean, et al., 1919).

Little information is at present available on the susceptibility of various *Nocardia* species to antibiotics.

Treponema pallidum is the cause of syphilis in man. It is a spiral organism with regular curves (6 to 14 in number), actively motile, with characteristic movements which assist in differentiating it from some saprophytic spirochetes. *T. pallidum* is best demonstrated by dark-field examination of exudates and by Levaditi or Fontana stains in tissues. There is, up to the present, no proof of successful cultivation of virulent *T. pallidum* on artificial media. *T. pallidum* is transmitted from man to man, who is the only natural host, although rabbits and monkeys can be infected and the organisms transmitted in them. Infection in man is usually contracted by sexual contact, and the majority of primary lesions are found on the genital organs; only about 10 per cent are found on the lips or tonsils and very rarely on other parts of the body (fingers, the professional primary of dentists and obstetricians). *T. pallidum* gains entrance through some minute break in the skin or mucous membranes, and some organisms may reach the regional lymph nodes in a matter of hours. After an incubation period of varying length (usually 18 to 21 days), the so-called primary lesion or chancre becomes apparent. The chancre can vary in size and shape from a barely visible superficial erosion to a bean-sized indurated lesion. Primaries of the lips or tonsils appear as irregular ulcers with a firm indurated base. These latter forms are the lesions with which the surgeon is often confronted, and the differential diagnosis on clinical grounds alone is not always possible. The regional lymph nodes are enlarged, firm, but freely movable, and the skin above them is unchanged. A dark-field examination of exudate or preferably of aspirated lymph will quickly establish the correct diagnosis. Lymph node aspiration is preferred if the lesion is in the mouth because some of the mouth spirochetes cannot be differentiated with cer-

tainty. A negative dark field does not exclude syphilis and several dark-field examinations should be made if the clinical features indicate syphilis. Before biopsies or other drastic measures are resorted to, serological tests should be performed over a period of time.

If the primary lesion is not recognized or in the female not suspected, syphilis enters the secondary stage after a period ranging from three to twelve weeks. This stage is characterized by general dissemination of *T. pallidum* throughout the body. The clinical picture of so-called "secondaries" is extremely varied and the student should refer to special textbooks. Rarely will the patient with secondary syphilis seek help from the surgeon, however, such lesions may appear during the postoperative phase of an unconnected ailment and the surgeon must be on guard. It has been estimated that approximately 25% of patients have spontaneous cures after the secondary stage, while a probably equal number enter the so-called latent phase, showing no signs or symptoms other than positive serological reactions.

A certain number of untreated or poorly treated cases, however, develop late complications, and these very frequently pose serious diagnostic problems. There is hardly an organ or tissue in the body which could not be affected; skin, muscle, bones and joints, the heart and blood vessels, and the central nervous system are most involved, but gummas of the stomach and liver also occur. In these late forms of syphilis the number of treponemata in the lesions is too small to be detected by our available methods, and the diagnosis can be made only by clinical observation, serological tests, histological and x-ray examinations. The symptomatology and differential diagnosis are discussed in the special chapters. The theoretical and practical details of the serological tests and the question of immunity are beyond the scope of this chapter.

Penicillin is at present the drug of choice for the treatment of syphilis. This anti-

biotic has altered the whole aspect of this disease and its control. Various treatment schemes are in use, the length of treatment varying from a few days to 2 or 3 weeks with an average dose of about 3,000,000-5,000,000 units. Over 90% of secondary and late cutaneous and gummatous lesions can be cured by penicillin. The treatment of cardiovascular and neurosyphilis is still a specialist's job.

Other spirochetal infections encountered by the surgeon are caused by *Borrelia vincenti*. This organism is much coarser and shows shallower spirals than *T. pallidum*. It can be readily stained with the usual aniline dyes. Vincent's angina, trench mouth, and many pulmonary abscesses show large numbers of *B. vincenti*, often in association with *Bacillus fusiformis* and streptococci. Infections of the skin and subcutaneous tissues by this group of organisms occur in wounds after human bites. Penicillin, either systemically or locally, is effective in the mouth lesions.

The presented outline is designated to cover common infectious agents and the disease processes caused by them as far as surgical practice in this country is concerned. A short survey of the "deep mycotic" infections, although relatively rare in Canada is appended.

FUNGUS INFECTIONS

Deep Mycotic Infections.—The fungi pathogenic for man belong to the *Fungi imperfecti*. The identification of fungi is based mainly on their morphological characters, the type of colony produced on various media and at special temperatures, and the microscopic appearance of the spores and mycelium. Fungus infections are much more common in tropical and subtropical climates. Because of their relative rarity in the temperate zones they are often not diagnosed, as neither physicians nor surgeons are very familiar with their symptomatology.

Sporotrichosis is caused by *Sporotrichum schenckii*. This fungus can be grown on the

common laboratory media and forms characteristic colonies on Sabouraud's agar. The isolation of the fungus is the only exact method of diagnosis. The fungus causes subacute or chronic granulomatous lesions usually on the exposed parts of the body. The primary lesion appears in the form of a rather uncharacteristic-looking abscess or ulcer which fails to heal. From this initial lesion, the fungus invades the lymphatics, and a very typical clinical picture develops: chronic lymphangitis with multiple subcutaneous abscesses along the lymph vessels. The abscesses rupture and chronic ulcers with no tendency of healing appear. These ulcers are usually secondarily infected with staphylococci or other organisms, and if only the superficial exudate is taken for cultures, the lesions are frequently diagnosed wrongly. Pus or exudate from unbroken lesions should be cultured. A skin test with heat-killed suspension of the fungus shows a positive reaction of the tuberculin type after the first week of disease.

The infection responds to treatment with iodides (Lugol's) orally, but such treatment has to be continued over a period of months.

Blastomycosis is a granulomatous disease of the skin or internal organs caused by *Blastomyces dermatitidis*. This fungus grows on the usual laboratory media and shows two types of growth, a yeastlike colony when grown at 37° C. and a moldlike form at room temperature. *B. dermatitidis* frequently infects the lungs and can be mistaken for tuberculosis. Spread via the blood vessels occurs and metastatic abscesses appear in the skin, the bones, liver, spleen and central nervous system. Primary infection of the skin consists of ulcers with a raised, irregular, papilliform border showing milium abscesses. The fungus can be isolated from the exudate and sputum. X-ray and iodides are still the best available treatment. Desensitization with vaccines may be necessary before iodides are given.

Coccidioidomycosis (Coccidioidal granuloma) is caused by *Coccidioides immitis*. The

occurrence of this organism appears to be confined to certain endemic areas (San Joaquin Valley in California, parts of Texas, and Arizona, etc.), where it has been found in the soil and is transmitted by dust. In the majority of cases infection of the lung takes place, primary infection of the skin through wounds is rare. The upper respiratory infection of the acute type manifests itself with the signs and symptoms of a bronchopneumonia with fever and chills and is known as San Joaquin fever, or Valley fever, which is generally a nonfatal, self-limited infection. Dissemination may occur and gives rise to the generalized form, known as coccidioidal granuloma which resembles tuberculosis very closely, both on clinical and x-ray findings. Lesions may appear anywhere in the body, and the mortality rate is very high. The diagnosis can be made by the cultivation of *C. immitis* from exudates or by the inoculation of mice and guinea pigs with pleural fluid, sputum, etc. A positive skin reaction of the tuberculin type to coccidioidin develops from 1 to 3 weeks after infection. Treatment of the systemic disseminated form offers little hope for success; x-ray, thymol, antimony and gentian violet, and specific vaccine therapy may be effective in individual cases.

Histoplasmosis is essentially a disease of the reticuloendothelial system caused by *Histoplasma capsulatum*. On cultures this organism grows in two morphological forms depending on the medium used and the temperature of incubation. On blood agar at 37° C and in tissues, *H. capsulatum* appears as small oval budding yeast cells which in tissue sections are found inside the cells. The organism enters the body through the lungs, the gastrointestinal tract, or skin abrasions. The clinical signs and symptoms may simulate a variety of diseases, carcinoma, Hodgkins', leukemia, tuberculosis, etc. The enlargement of lymph nodes is usually an outstanding feature. The diagnosis depends on the isolation of *H. capsulatum* from the peripheral blood, the bone marrow, lymph

nodes or skin ulcers, or on the demonstration of the organism in histological sections. The skin test with histoplasmin may be negative even in active infection; however, positive skin tests have been obtained in large series in cases with calcification of the lungs and negative tuberculin test. This is taken as evidence that histoplasmosis as a mild self-limited disease is much more common than was previously suspected. The prognosis in progressive generalized histoplasmosis is generally hopeless; no effective drug is as yet available.

Cryptococcosis (Torulosis).—This fungus infection due to *Cryptococcus neoformans* shows a decided preference for the central nervous system. *C. neoformans* is a budding yeastlike organism showing both in cultures and tissues or exudates a large capsule. *C. neoformans* can be easily cultured on ordinary laboratory media. The central nervous system is the most frequently affected organ either primarily or secondarily from lung or skin lesions. Clinically, the infections resemble tuberculous meningitis, brain abscess, brain tumor or syphilis. The cutaneous lesions appear in the form of subcutaneous abscesses or tumors which break through the skin and form ulcers. Generalized spread from the skin lesions almost invariably involves the central nervous system as well as the lungs, liver, spleen, etc. The diagnosis is made by the demonstration of the fungus in exudates, by cultural isolation or animal inoculation (mice). A drop of spinal fluid or pus or other exudates, suspected of containing *C. neoformans* should be mixed with a drop of India ink on a slide under a cover slip. When viewed under the microscope the fungus appears as a round, budding cell surrounded by a large capsule. No generally effective treatment of cryptococcosis is known. The central nervous form is almost invariably fatal. Improvement in some cases has been reported after the use of iodides, sulfa drugs, and x-ray, but it is by no means consistent.

Other fungi may occasionally cause lesions which have to be differentiated from other infections. For detailed information on fungus infections of medical importance, the student should consult special textbooks (Lewis and Hopper, 1948) (Conant, 1918).

APPENDIX

In the diagnosis of infections the proper taking of specimens is of the utmost importance. Even the best bacteriological laboratory can only isolate the flora in the specimen received. Certain principles have to be kept in mind to make a quick and accurate diagnosis possible, and the physician and surgeon have to contribute their part.

1. The introduction of contaminating organisms must be prevented; instruments used for taking the specimens and the containers must be sterile.

2. Specimens should be taken from the freshest lesions, and the site of progressive infection, e.g., from the edges of ulcers, from unopened abscesses, rather than broken ones whenever possible.

3. Sufficient material should be collected, as far as feasible, if various examinations are required or when the chances of finding the causative organism increase (up to a point) with the amount of material available (e.g., 24-hour specimens of urine for the demonstration of tubercle bacilli).

4. Information on the clinical condition of the patient and on any treatment received has to be given to the laboratory to aid in the selection of suitable media and techniques; whenever possible specimens should be collected before treatment is started. In problem cases the collaboration of the surgeon and laboratory chief is essential for results and their evaluation.

5. Last but not least, specimens should reach the laboratory as soon as possible after they have been obtained to prevent drying or other injuries to fastidious organisms. Details on the taking of blood cultures and

specimens from various regions can be found in textbooks (Murray and Kalz, 1949) (Marshall, et al, 1947).

REFERENCES

- Alexander, H. E., and Leidy, G.: The Present Status of Treatment for Influenzal Meningitis, *Am. J. Med.* 2: 457-466, 1947.
- Avery, O. T.: The Role of Specific Carbohydrates in Pneumococcus Infection and Immunity, *Ann. Int. Med.* 6: 1-9, 1932.
- Cairns, Hugh, et al.: Tuberculous Meningitis, *J. A. M. A.* 144: 92-96, 1950.
- Chain, E., et al.: Penicillin as Chemotherapeutic Agent, *Lancet*, 2: 226-228, 1940.
- Conant, N. F.: In Dubos, R. J., ed.; *Bacterial and Mycotic Infections of Man*, Philadelphia, 1948, J. B. Lippincott Company, pp. 588-627.
- Coventry, M. B., et al.: Infection of Hip by *Brucella suis*, *J. A. M. A.* 141: 320-325, 1949.
- Cutino, Y. T., and McCabe, A. M.: Pure Granulomatous Nocardiosis; a New Fungus Disease Distinguished by Intra-cellular Parasitism, *Am. J. Path.* 25: 1-47, 1949.
- Dowling, H. F., et al.: Treatment of Pneumococcal Meningitis With Massive Doses of Synthetic Penicillin, *Am. J. M. Sc.* 217: 149-156, 1949.
- Dubos, R. J., and Middlebrook, G.: Media for Tubercle Bacilli, *Am. Rev. Tuberc.* 56: 334-345, 1947.
- Fleming, A.: On Antibacterial Action of Cultures of Penicillin With Special Reference to Their Use in Isolation of *B. influenzae*, *Brit. J. Exper. Path.* 10: 226-236, 1929.
- Foshay, L.: Tularemia: A Summary of Certain Aspects of the Disease Including Methods for Early Diagnosis and the Results of Serum Treatment in 600 Patients, *Medicine* 19: 1-83, 1940.
- Griffith, F.: The Serological Classification of *Streptococcus Pyogenes*, *J. Hyg.* 34: 542-584, 1933.
- Hewitt, W. L., and Williams, B.: Chloromycetin (Chloramphenicol) in the Treatment of Infections, *New Eng. J. Med.* 242: 119-127, 1950.
- Huddleson, I. F., et al.: *Brucellosis in Man and Animals*, ed. 2, New York, The Commonwealth Fund, pp. 149-244, 1943.
- Johansson, K. R., and Sarles, W. B.: Some Considerations of the Biological Importance of Intestinal Micro-organisms, *Bact. Rev.* 13: 25-45, 1949.
- Julianelle, L. A.: A Biological Classification of *Encapsulatus Pneumoniae* (Friedlander's Bacillus), *J. Exper. Med.* 44: 113-128, 1926.
- Kirby, W. M. M., and McNaught, J. B.: Actinomycosis Due to *Nocardia asteroides*, *Arch. Int. Med.* 78: 578, 1946.
- Lancefield, R. C.: The Antigenic Complex of *Streptococcus hemolyticus*, *J. Exper. Med.* 47: 91-103, 481, 1928.
- Lewis, G. M., and Hopper, M. E.: *An Introduction to Medical Mycology*, ed. 3, Chicago, 1948, The Year Book Publishers, Inc.
- Long, P. H.: The Clinical Use of Antibiotics *M. Clin. North America* 34: 307-318, 1950.

- Marshall, M. S.: *Applied Medical Bacteriology*, Philadelphia, 1917, Lea & Febiger.
- Meleney, F. L.: *Zinc Peroxide in the Treatment of "..."*, 1919, W. H. Saunders Company.
- Murray, E. G. D., and Denton, G. D.: Plaster of Paris as Source of Infection in Tetanus and Gas Gangrene, *Canad. M. A. J.* 60: 1-4, 1919.
- Murray, E. G. D., and Kalz, G.: In Dubos, R. J., ed: *Bacterial and Mycotic Infections*, Philadelphia, 1918, J. B. Lippincott Company, pp 701-739.
- MacLennan, J. D.: Anaerobic Infections of War Wounds in the Middle East, *Lancet* 2: 91, 1913.
- McVay, L. V., Jr., Guthrie, F., and Sprunt, D.: Septicemia Due to *Bacteroides*, Aureomycin Hydrochloride Therapy in Two Cases Due to *Bacteroides funduliformis*, *J. A. M. A.* 140: 1150-1152, 1949.
- Nichols, D. R., and Herrell, W. E.: Penicillin in Treatment of Actinomycosis, *J. Lab. & Clin. Med.* 33: 521, 1948.
- Pfuetze, K. H., and Pyle, M. M.: Streptomycin in the Treatment of Tuberculosis, *J. A. M. A.* 139: 634-639, 1949.
- Pratt, R., and Dufrenoy, J.: *Antibiotics*, Philadelphia, 1949, J. B. Lippincott Company.
- Reed, G. B.: In Dubos, R. J., ed: *Bacterial and Mycotic Infections of Man*, Philadelphia, 1918, J. B. Lippincott Company, pp 355-369.
- Rich, A. R.: *The Pathogenesis of Tuberculosis*, Springfield, Ill., 1911, Charles C Thomas.
- Schipper, G. J.: Unusual Pathogenicity of *Pasteurella multocida* Isolated From the Throats of Common Wild Rats, *Bull. Johns Hopkins Hosp.* 81: 333-356, 1917.
- Seibert, F. B., et al.: Purified Protein Derivative. A Standardized Tuberculin for Uniformity in Diagnosis and Epidemiology, *Am. Rev. Tuberc.* 30: 705-768, 1934.
- Sherman, J. M.: The Streptococci, *Bact. Rev.* 1: 3-97, 1937.
- Smith, L. D.: Clostridia in Gas Gangrene, *Bact. Rev.* 13: 233-254, 1949.
- Symposium on Terramycin: *Ann. New York Acad. Sc.* 53: 221-460, 1950.
- Waksman, S. A., Bugie, E., and Schatz, A.: Isolation of Antibiotic Substances From Soil Microorganisms, With Special Reference to Streptomycin and Streptomycin, *Proc. Staff Meet., Mayo Clin.* 19: 537-548, 1944.
- Waksman, S. A., and Lechevalier, H. A.: Neomycin, a New Antibiotic Active Against Streptomycin-Resistant Bacteria, Including Tuberculosis Organisms, *Science* 109: 305-307, 1949.
- Wood, W. B., Smith, M. R., and Watson, B.: Studies on the Mechanism of Recovery in Pneumococcal Pneumonia, *J. Exper. Med.* 84: 387, 1916.
- Woodward, T. E., et al.: Aureomycin in Treatment of Experimental and Human Tularemia, *J. A. M. A.* 139: 830-832, 1949.
- Zintel, H. A., et al.: Absorption, Distribution, Excretion and Toxicity of Bacitracin in Man, *Am. J. M. Sc.* 218: 439-445, 1949.

CHAPTER IV

SHOCK AND BLOOD TRANSFUSION

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SHOCK

No subject in surgery is of greater importance than shock. Despite a better understanding of its causes and treatment gained from the many investigations of the problem and by the development of blood banks and transfusion services during the last ten years, shock still remains a dreaded complication of surgical operations and trauma.

Shock, also known as *collapse* or *peripheral circulatory failure*, may be defined as the syndrome characterized by subnormal temperature, pallor, cyanosis, sweating, cold extremities, and low blood pressure due to a reduction in blood volume usually the result of hemorrhage.

Until a few years ago, shock perhaps even excelled eclampsia as the "Disease of Theorists." There was general agreement that the main factor in shock was an acute reduction in blood volume, but there was, however, a difference of opinion on the cause of the decrease in blood volume.

Theories.—The principal theories and those on which present-day management of shock depends are

1. *Neurogenic*. Nervous impulses originating from tissue damaged by trauma or at operation cause a dilatation of the vascular system leading to a reduction in effective blood volume.

2. *Toxic*. Noxious substances liberated by damaged tissue increase the permeability of capillaries leading to loss of plasma from the circulation, reduction in blood volume and hemoconcentration, i.e., a relative increase in the cellular elements of the blood.

3. *Hemorrhagic*. The reduction in blood volume is due to hemorrhage resulting from trauma, operation or occurring spontaneously.

These conflicting theories have recently been resolved and the following concepts are those now generally held. *Primary*, or *neurogenic*, shock is due to a dilatation of the vascular system. *Secondary*, or *oligemic*, shock, also known as *traumatic* and *surgical shock*, is due to a reduction in blood volume resulting from fluid loss. The fluid lost may be water and electrolytes in dehydration, plasma as in burns or whole blood from hemorrhage.

The terms *primary* and *secondary* are not used in respect of the time of appearance of shock. They are essentially different conditions. Of the two, the latter is the more important and the account that follows is therefore mainly concerned with secondary shock.

Pathological Physiology.—In shock due to fluid loss there is a *decreased cardiac output* resulting from inadequate filling of the ventricles because of the diminished venous return. *Anoxia* may result from the circulatory failure and give rise to an *acidosis*. An impaired renal function of a transient nature may occur or the more severe renal damage of *lower nephron nephrosis* may supervene. *Liver damage* as demonstrated by various liver function tests has been shown to accompany shock. The *brain* is very susceptible to anoxia and if shock is prolonged or severe, there may occur after a period of apparent recovery, *coma*, *convulsions*, and *death*.

There are also compensatory changes consisting of *vasoconstriction* and *hemodilution*.

The effect of the former is to reduce the size of the vascular bed by selective vasoconstriction in the skin and kidneys. It has been estimated that approximately 500 c.c. of blood normally contained in the vessels of the skin are thereby made available for vital organs such as the brain. Vasoconstriction in the vessels of the kidneys may have a damaging effect as mentioned above. Vasoconstriction is also important in that, by maintaining blood pressure at or near normal levels, it may mask severe hemorrhage and impending shock. In severe or untreated cases of shock, the mechanism ultimately fails.

A less effective compensatory mechanism is hemodilution. By this means the reduced blood volume is partially restored by passage of water and electrolytes from tissue spaces into the blood stream. Hemodilution may not occur in dehydration or in severe anemia. Vasoconstriction and hemodilution cannot be relied upon, therefore, to maintain an adequate blood volume in cases in which hemorrhage has been severe or prolonged.

Diagnosis.—The clinical features of shock—the signs and symptoms such as restlessness, subnormal temperature, rapid pulse, pallor, sweating, cold extremities and low blood pressure, the metabolic and other changes already described—are those of the fully developed state. It is the *early recognition* of the causes of reduction in blood volume that should be the aim of diagnosis in order to prevent shock and its grave effects.

The most common cause of reduction in blood volume is *hemorrhage*. The hemorrhage may be either external or internal. It may be due to trauma; it may occur during or following operation; or it may be spontaneous, as, for example, from a peptic ulcer, ruptured ectopic pregnancy, thrombocytopenic purpura.

The large amounts of blood that may be lost in traumatic injuries or at operation have been shown in numerous studies. It

can be anticipated that wounds which are caused by violent force and which result in extensive injury will be accompanied by shock.

Loss of blood into the soft tissues, body cavities or gastrointestinal tract is difficult to evaluate. The swelling of soft tissues, or the presence of dullness on percussion over body cavities is useful as an indication of the extent of hemorrhage.

Once shock has developed, the only reliable clinical guide is the level of the blood pressure. (In individuals with hypertensive disease, the blood pressure may not fall below the normal range.) The pulse rate is not always increased. Blood studies such as hemoglobin or hematocrit determinations are of no use inasmuch as they do not reflect the changes in blood volume in hemorrhage. Blood volume estimations may be valuable in complicated cases, especially if a preoperative determination has been made.

Reduction of blood volume from *loss of plasma* most commonly occurs in cases of burns. Plasma is lost from damaged capillaries in and adjacent to the burned areas. The loss of plasma leads to hemoconcentration. In intestinal obstruction, especially when accompanied by local circulatory stasis as in mesenteric thrombosis, there may be a diffusion of plasma into the peritoneal cavity and bowel lumen with a resultant reduction in blood volume. The crush syndrome follows compression injuries to the extremities. When the compression is released, plasma is lost into the damaged tissues, and shock develops.

In conditions leading to *dehydration* such as excessive vomiting, diarrhea, gastrointestinal fistulas, there is a reduction in blood volume due to loss of water and electrolytes from the blood.

Although of no use in hemorrhagic shock, the determination of the degree of hemoconcentration by hemoglobin or hematocrit studies is of great value in estimating the degree of reduction of blood volume from loss of plasma (or in dehydration) and in

determining the amount of plasma (or saline) to be infused.

Prophylaxis.—Patients in shock withstand operative procedures poorly. As a rule surgical measures should not be undertaken until shock has been relieved. When immediate operation is imperative to control hemorrhage or for other urgent reasons, transfusion should be started at once and continued throughout the operation.

crease in both the volume and particularly the oxygen-carrying capacity of the blood must be restored by transfusions of whole blood before major operations are performed on such patients. The usual practice is to give 500 to 1,000 c.c. of blood, the amount depending on the degree of anemia, on each of several successive days immediately prior to the day of operation. Red cell suspensions as described later may also be used.

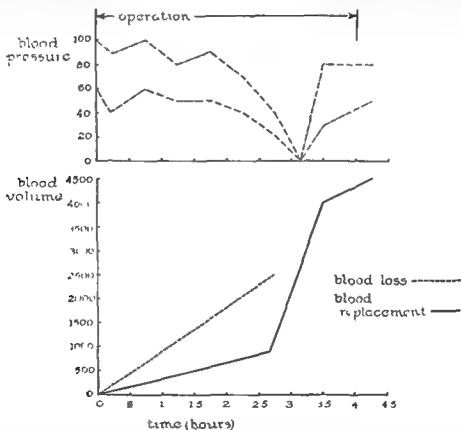


Fig 1—Illustrates: (a) the large amount of blood that may be lost at operation, in this case a pneumonectomy for pulmonary tuberculosis, (b) the development of shock during operation because of failure to maintain blood replacement *pari passu* with blood loss which was measured by the Wangenstein method, and (c) the proper treatment of shock by immediate, rapid and adequate replacement of blood.

In many patients with chronic disease there is often an associated anemia due to various causes including malignancy, infection, and malnutrition. In such patients there is usually a reduction in total blood volume because the reduction in the red cell mass may be only partially compensated for by an increased plasma volume. The de-

The determination of prothrombin and the use of vitamin K and fresh blood transfusion will prevent hemorrhage due to *hypoprothrombinemia*.

The choice of an anesthetic demands great care when shock is either present or anticipated. Oxygen should be given continuously during anesthesia in all cases of

shock to avoid anoxia. It is the responsibility of the anesthetist to watch for the early signs of shock in all operations, looking particularly for falling blood pressure. Blood pressure readings should be made repeatedly during the operation and at frequent intervals after the patient's return to the ward.

Careful *surgical technique* both in the gentle handling of tissue and the use of hemostasis is important in minimizing hemorrhage.

mediate *re-operation* for control of hemorrhage in all patients who develop shock following operation and in whom the blood pressure cannot be restored or maintained by transfusions.

Treatment

The treatment of shock consists principally in the replacement of the fluid lost and, to be discussed later, the use of vasoconstrictor drugs and hormones in cases in

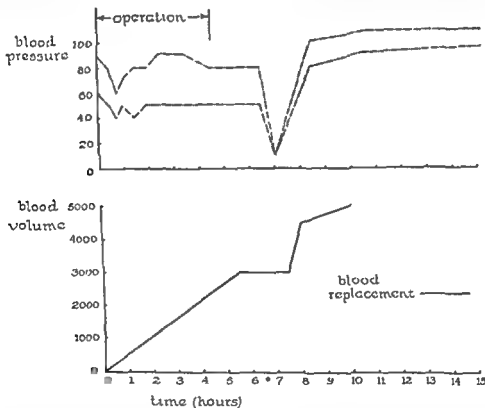


Fig. 2—Illustrates: (a) the large amount of blood that may be lost during surgical operation, in this case removal of mediastinal tumor; (b) the development of shock several hours after the end of the operation; and (c) the proper treatment of shock by immediate rapid and adequate blood transfusion

Shock may manifest itself not only during or at the end of operation but also at any time after the operation; even hours later. When shock occurs several hours after the end of an operation, it may be due to hemorrhage from a loose ligature. In such cases, transfusions will have only a temporary effect in restoring the blood pressure. Serious consideration must be given, therefore, to im-

which vasodilatation is a factor. In *hemorrhagic shock* there is no substitute for *whole blood*. Plasma transfusion, although it restores blood volume in this condition, does not improve the reduced oxygen-carrying capacity of the blood. The use of plasma or plasma substitutes in shock from hemorrhage should be confined to the emergency measure of restoring or maintaining blood

volume during the period of delay necessary to the starting of a whole blood transfusion or in supplementing blood transfusion if supplies of blood are limited.

Just as important as knowing *what to give* is knowing *how to give it*. In shock from hemorrhage, blood must be given *immediately*, it must be given *rapidly* and often in *large quantities*, of the order of 5 to 10 liters. These three aspects of the treatment of shock cannot be overemphasized. They are determined not by one's impression of the amount of blood lost (which is usually an underestimation), or by the large amount which may have already been administered, but only by the response of the patient to transfusion as indicated by the level of the arterial blood pressure.

taching the pump of a blood pressure apparatus to the air inlet tube of the transfusion bottle and applying pressure, care being taken to prevent the entrance of air into the flow of blood. In the severely shocked patient, with cold extremities, the peripheral veins are usually contracted. Dilatation of the veins may be induced by the *application of heat* to the extremity to be used for transfusion. Since, however, time is of the essence in the treatment of shock, at the first indication that the collapsed state of the veins may delay a successful venipuncture, a "cut-down" should be started at once.

The technique of the "cut-down" is as follows. The site usually chosen is the ankle, although any of the larger veins in the arm may be used. If the ankle is chosen,

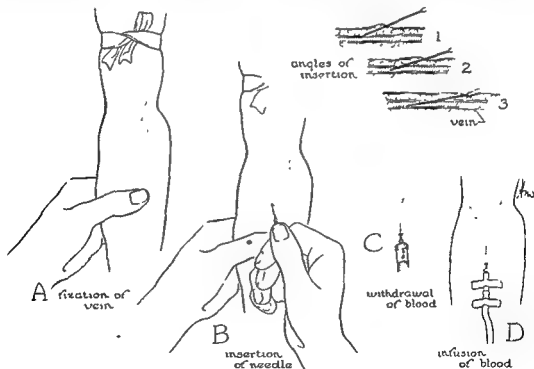


Fig 3—Technique of venipuncture

Technique of Transfusion.—There are several devices frequently used for the rapid restoration and, with continuing blood loss, the maintenance of normal blood volume. Blood may be transfused into *several veins simultaneously*. It may be *forced in* by at-

the vein may be found lying immediately above and in front of the medial malleolus. Under local anesthesia the vein is exposed with blunt dissection. It is separated from the surrounding tissues by placing the handle of a scalpel or other flat instrument between

the under surface of the vein and the skin. Two ligatures are then drawn between the vein and the skin; one is placed distally and tied around the vein; the proximal ligature is left untied. A small incision is made in the anterior wall of the vein between the two ligatures with a pair of scissors or other sharp-pointed instrument. A Lindemann

fill rapidly the renal, coronary and cerebral vessels, *simultaneously with the restoration of blood volume.*

In burns, plasma should be supplemented with whole blood in order to correct the early anemia which is a constant feature of severe burns. In certain cases of intestinal obstruction, peritonitis and abdominal wounds, plasma or whole blood transfusions may not be effective, presumably because of irreversible dilatation of the small blood vessels due to the action of toxic products of infection.

Dehydration must be treated with saline and not glucose, since it is water and electrolytes that have to be replaced. In intestinal obstruction saline as well as plasma must be infused to replace the electrolytes lost in vomiting.

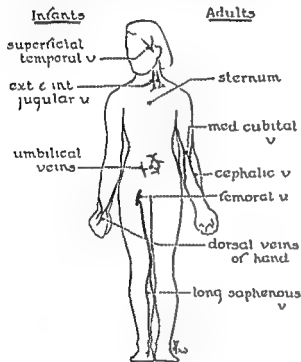


Fig 4—Routes for transfusion

needle or cannula is then inserted proximally for some distance into the vein and tied securely in place by means of the second ligature. The needle or cannula may be immediately connected to a transfusion set or kept patent by the attachment of a slow-drip intravenous solution of saline.

Intra-arterial transfusion has recently been introduced as a method to be used in profuse hemorrhage or profound shock when restoration of the blood volume by the intravenous route is ineffective. The transfusion is usually administered via one of the arteries of the hand,* most often the radial. By means of pressure, blood may be forced retrograde into the arterial system so as to

*In a right-handed patient, intra-arterial transfusion should be given in the left hand.

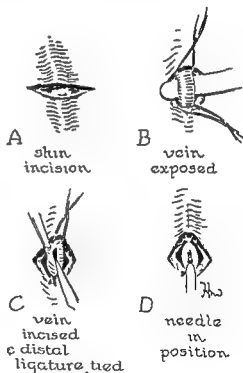


Fig. 5.—Technique of the cut-down.

Neurogenic shock may occur following spinal anesthesia and after the second stage of sympathectomy as a result of the interference with the vasoconstrictor nerve supply to the lower parts of the body. Primary shock may be one of the manifestations of a

severe transfusion reaction. The removal of a pheochromocytoma, or a diseased kidney causing hypertension may also be followed by shock due to vasodilatation resulting from the sudden withdrawal of the vasopressor agents, epinephrine or renal pressor substances.

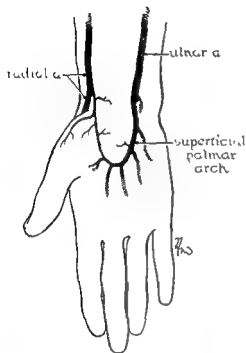


Fig 6—Arteries of the hand

Shock of similar origin may occur in operations on patients with adrenal cortical insufficiency. Adrenal cortical hormone plays a role in the maintenance of normal vasomotor tonus. The use of ACTH and cortisone, depending on whether the adrenal cortical failure is secondary to disease of the pituitary gland or primarily adrenal in origin, will minimize neurogenic shock in operations on such patients.

In shock due to causes other than a reduction in blood volume, i.e., failure of the heart itself, pericardial tamponade, infection, and pulmonary embolism, transfusion is either ineffective or contraindicated.

Adjuvant Measures.—Although transfusion is the main measure in the therapy of secondary shock there are other adjuvant

measures which have a valuable place in its treatment.

1 The control of hemorrhage is obviously of utmost importance.

2 Vasoconstrictor drugs are valuable agents in counteracting vasodilatation and in restoring the reduction in effective blood volume in cases of neurogenic shock. The use of these drugs is unsatisfactory, however, in cases of secondary shock. ACTH and cortisone are useful in the prevention and treatment of shock only in conditions of adrenocortical insufficiency.

3 Immobilization of fractures is an important measure, preventing further trauma at the fracture site.

4 The administration of oxygen is beneficial in cases complicated by cardiopulmonary disease or injury, and during anesthesia.

5 Apprehension, pain, and fatigue must be allayed because they may induce vasodilatation. Morphine, judiciously used, is the drug of choice.

6. Body temperature should be maintained but the excessive application of heat, an effective vasodilator, by relaxing compensatory vasoconstriction may precipitate shock.

7 Because of vasomotor instability, the patient who is bleeding or already in shock should be moved as little as possible except for posturing in the head-low position. Elevation of the foot of the bed in patients with a moderate reduction in blood volume causes a definite increase in blood pressure indicating that the amount of blood shifted from the lower parts of the body to its center improves the vasomotor status. The shock position may be inadvisable in patients with cerebral or cardiopulmonary complications.

In conclusion, it is worth while to repeat that blood must be given as soon as possible after hemorrhage, it must be given rapidly and in adequate amounts. The view that blood transfusion by raising the blood pressure may increase, or cause a resumption of bleeding, is entirely unfounded and if transfusion is for this reason withheld the results

may be fatal. In cases of massive hemorrhage requiring large amounts of blood to restore blood pressure to normal, inadequate amounts may be given for fear of overloading the circulation. Such fears are likewise unfounded if the level of the blood pressure is used as the guide for determining the amount of blood to be used. Only after the

may then be continued at a slower rate. Signs of increasing the blood volume beyond normal may be found by inspecting the veins for fullness or by the more accurate method of measurement of venous pressure. More deaths result from transfusion given "too little and too late" than from circulatory overloading.

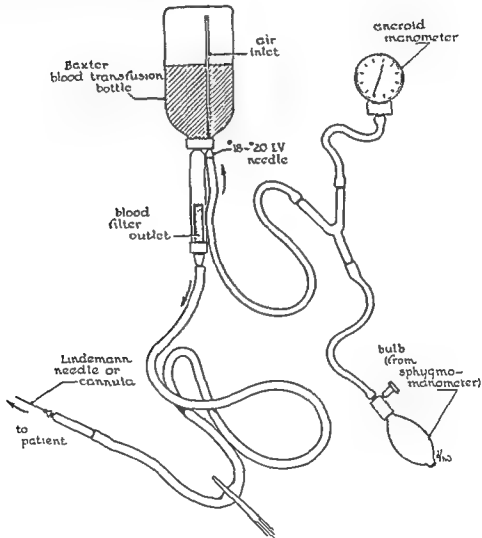


Fig 7—Blood transfusion apparatus with pressure connection for rapid intravenous or intra-arterial transfusion.

blood volume and blood pressure have been restored to normal is it likely that further transfusion will overload the circulation. When the normal blood volume as reflected by a normal blood pressure has been achieved and maintained, the need for rapid transfusion no longer exists. The transfusion

The treatment of shock because of its emergency nature has been compared with the treatment of diabetic coma in that it is not enough to start treatment, write orders, and return later to see how the patient is progressing. There is probably no other condition that demands such resourcefulness,

close supervision, and sense of urgency during the entire period of treatment. The effects of anoxia are so serious that in profound shock the period during which transfusion may be effective is often measured only in minutes, almost never in hours.

BLOOD TRANSFUSION

The recent advances in the recognition and treatment of shock and the development of blood banks and transfusion services have contributed greatly to the progress of surgery. They have become important factors both in the performance of operations and in the recovery from injuries in which the amount of blood lost may be considerable. Shock has been discussed in the preceding section.

Blood transfusion may be considered from five main aspects:

- 1 Biochemical
2. Immunological
- 3 Therapeutic
- 4 Reactions
- 5 The blood bank and transfusion services

Biochemical Aspects.—The constituents of whole blood which are of chief importance in transfusion therapy are the erythrocytes and the plasma. The solutions used for the preservation of blood have been designed for the best *preservation of the erythrocytes* without too great dilution of the whole blood mixture. The solution includes sodium citrate as anticoagulant and dextrose as preservative. The proportion of whole blood to solution of anticoagulant and preservative in the average bottle of blood is approximately three parts whole blood to one part diluent. As an additional preservative measure, blood must be stored at a temperature of 4° C. Freezing and thawing destroy the erythrocytes.

Under the ordinary storage conditions of the blood bank, blood may be used up to three weeks following donation. Maximum benefit is obtained by the transfusion of fresh

blood. The rate of loss of viability of erythrocytes in preserved blood is approximately 1% of the cells per day, but after three weeks they deteriorate more rapidly. Blood which has not been used by the end of this period is set aside to permit the sedimentation of the cellular elements. The supernatant plasma is then drawn off, and stored. The cells are discarded.

Plasma can now be separated into various components by fractional precipitation of the plasma proteins with alcohol at low temperatures. The plasma may be divided into five main fractions. Fraction I contains fibrinogen which is used in surgery as fibrin film or as fibrin foam impregnated with thrombin to promote coagulation. It also contains an antihemophilic protein which causes a decrease in the clotting time of hemophiliacs. Fractions II and III contain the alpha, beta and gamma globulins which include the various antibodies, hemagglutinins and prothrombin. One of the gamma globulin fractions has proved valuable in the prophylaxis of measles. Fraction IV contains miscellaneous substances of little clinical significance to date. Fraction V is an important one as it contains *albumin*, the most important constituent of plasma from the standpoint of colloid osmotic properties and for whose beneficial effects most transfusions of plasma are given. It is by virtue of the high osmotic value of albumin that plasma is used for the prevention and treatment of shock from hemorrhage when whole blood is not available. The condition referred to as hypoproteinemia implies hypoalbuminemia. It is the ready utilizability of albumin which makes plasma valuable in the treatment of cirrhosis of the liver, nephrosis, and diseases of the gastrointestinal tract in which albumin either is not synthesized at the normal rate or is lost from the circulation in large amounts. Albumin has also been used because of its high osmotic value in the treatment of cerebral edema.

Various *plasma substitutes* have been tried since acacia was first introduced during

World War I. Gelatin, dextran, isinglass, pectin, bovine albumin, polyvinyl pyrrolidone and other synthetic macromolecular materials have been used. Some of these substances are toxic; bovine albumin is antigenic; acacia and pectin are deposited in the reticulo-endothelial system. Gelatin and dextran are relatively safe substitutes for plasma and superior to crystalloid solutions in the emergency treatment of shock.* They are, however, neither the physiologic nor the metabolic equivalent of plasma when used in the conditions for which plasma is indicated, such as burns and hypoproteinemia.

Platelets and leukocytes disintegrate rapidly in preserved blood; the majority of the former disappear within several hours, the latter within two or three days of storage. Prothrombin is fairly well maintained in preserved blood and does not show a significant decrease until after the first week of storage. For the treatment of the purpuras and hypoprothrombinemia it is advisable to use fresh blood.

Plasma may be stored in the frozen or dried state as well as in the liquid form. Storage of plasma in the liquid state is the simplest and most economical method. Fibrinogen tends gradually to be precipitated during storage. The globulins and albumin are not altered during the usual periods of storage in the frozen or dried state.

The use of *red cell suspensions* or "*packed cells*" as they are commonly called has proved valuable in the treatment of chronic anemia in patients with cardiac or renal disease in whom the volume of fluid administered must be restricted. This procedure also conserves plasma for, except in cases of shock or hypoproteinemia, whole blood is not usually needed in the treatment of chronic anemia. The method of preparation consists in allowing the red cells to sediment spontaneously or with the aid of centrifuging, drawing off the supernatant plasma, and transferring the cells to a separate container. If the blood has been collected in one of the

dextrose preservative solutions, the cells may be stored in the sedimented state up to two weeks. When they are to be used they may be re-suspended in saline. After the appropriate grouping and cross-matching, the red cell suspension may be administered in the same manner as whole blood.

The preparation of plasma and red cell suspensions demands the most *careful technique* and *sterile precautions* since these materials, which are excellent bacterial culture media, may be stored for a considerable period before being used. It is important to note that some pathogenic organisms whose toxins are lethal may continue to multiply in contaminated plasma even when stored at 4° C. If a transfusion for any reason has to be stopped, the same bottle of whole blood, plasma, red cell suspension or any other intravenous solution should not be used again if more than an hour has elapsed between the first attempt and the next because of the danger of contamination.

Whole blood, plasma or red cell suspension must always be administered with an intravenous set containing a *fine mesh filter* to remove particulate matter or clots that may have formed.

Immunological Aspects.—The immunological factors of clinical importance in blood transfusion are:

1. The agglutinogens A and B.
2. The Rh and Hr factors.
3. Other rare factors.

Of equal concern are the corresponding antibodies, the agglutinins anti-A, anti-B, anti-Rh, anti-Hr, etc.

The Landsteiner (or ABO) blood groups. All individuals may be divided into four groups depending on whether their erythrocytes contain either of the agglutinogens A or B, both, or neither of them. Agglutinins are present in the serum of an individual corresponding to the agglutinin which is absent from the erythrocytes of that individual as illustrated in Table II.

In every transfusion it is the donor's erythrocytes which must be compatible with the

*Polyvinyl pyrrolidone, more commonly known as PVP, is also a satisfactory substitute.

TABLE II

INTERNATIONAL (LANDSTEINER) NOMENCLATURE

| NAME OF BLOOD GROUP | AGGLUTINOGEN IN ERYTHROCYTES | AGGLUTININ IN SERUM |
|---------------------------|------------------------------------|------------------------|
| AB | AB | Neither |
| A | A | anti-B (beta) |
| B | B | anti-A (alpha) |
| O | Neither | anti-A and anti-B |

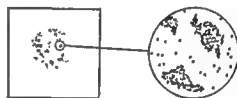
recipient's, i.e., not agglutinated (clumped), by the agglutinins in the recipient's serum. As indicated in Table III Group O blood may be given to individuals of any group because O blood contains neither agglutinogen A nor B. Persons belonging to Group AB may receive blood of any group because their serum does not contain either agglutinin anti-A or anti-B. With these two exceptions donor and recipient blood must belong to the same group to avoid agglutination, hemolysis, and the serious consequences of an incompatible transfusion reaction.

Laboratory Technique—Students and internes should be familiar with the technique of grouping and cross-matching blood because of the frequent urgent need for transfusion in the prevention and treatment of shock. The test for determining to which of the four blood groups a patient or donor belongs is performed as follows:

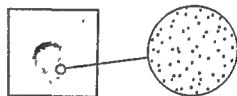
Two drops of citrated blood are added to approximately 5 c.c. of normal saline. A drop of the saline cell suspension is added to each of two serological tubes. To one tube add a drop of anti-A grouping serum, to the other tube add a drop of anti-B grouping serum. Centrifuge at 1000 rpm for two

minutes. Examine for agglutination with the aid of a microscope. Reference to Table III will show how any unknown blood can be typed by noting the reaction of the erythrocytes with the anti-A and anti-B serums.

The technique of cross-matching is as follows. To a serological tube marked "major" add a drop of a saline suspension of donor's cells and a drop of recipient's serum or plasma. To another tube marked "minor" add a drop of a saline suspension of recipient's cells and a drop of donor's plasma. Centrifuge and examine for agglutination.



positive result
~agglutination~



negative result
~no agglutination~

Fig. 8—Blood grouping and cross-matching as viewed through the microscope. High power on right, low power on left.

Clumping of the cells in the *major cross-match* indicates that the bloods are incompatible and that the donor blood must not be given.

Agglutination in the *minor cross-match* may occur if (1) Group O donor blood is being tested against the blood of A, B or AB recipient. (2) Group AB recipient blood is being tested against the blood of an A, B or O donor. Only in these two instances may agglutination in the *minor cross-match* be

TABLE III

INTERACTION OF THE FOUR BLOOD GROUPS

| | AB SERUM (NO AGGLU- TININS) | A SERUM (ANTI-B AGGLU- TININS) | B SERUM (ANTI-A AGGLU- TININS) | O SERUM (ANTI-A AND B AGGLU- TININS) |
|----|---|---|---|---|
| AB | - | + | + | + |
| A | - | - | + | + |
| B | - | + | - | + |
| O | - | - | - | - |

+ = agglutination
- = no agglutination

disregarded because otherwise it indicates that the donor and recipient blood are incompatible.

The Rh Blood Groups.—There are three Rh factors, Rh_a , rh' and rh'' . The clinical importance of the various Rh factors is directly related to their antigenicity, i.e., their power to elicit antibody response. Only those individuals whose erythrocytes contain the strongly antigenic agglutinin Rh_a are said to be *Rh positive*.

Individuals in the population who are *Rh negative* have in their erythrocytes, however, an agglutinin known as Hr . There is thus an inverse relationship between the presence and absence of Rh and Hr factors. Rh negative individuals are Hr positive and vice versa. There are also three Hr factors, Hr_a , Hr' and Hr'' . Like rh' and rh'' the three Hr factors are of less clinical significance because they are weak antigens. Hr' may, however, in rare instances act similarly to Rh_a in producing *isoimmunization* or *isosenitization* presently to be described. Other more rare factors related to the Rh factor which may also cause isoimmunization have been discovered.

Although the anti-Rh agglutinins are not normally present in the serum, they may be acquired during *pregnancy* or after *transfusion of blood* of a different Rh type from that of the recipient. If a mother is Rh negative and the father Rh positive, the fetus may be Rh positive. The Rh factor present in the erythrocytes of the fetus and foreign to the mother may act as an antigenic stimulus with the consequent production of anti-Rh agglutinins by the mother. This process is known as *isoimmunization*.

The anti-Rh agglutinins (Rh antibodies) combine with the Rh agglutinogens of the fetal erythrocytes and cause hemolysis, a condition known as *hemolytic disease of the newborn* or *erythroblastosis fetalis*. Nothing happens to the mother because there are no Rh agglutinogens in her erythrocytes to combine with the antibodies in her serum. If,

however, she receives a transfusion of Rh positive blood during pregnancy or in the puerperium, the transfused erythrocytes combine with the Rh antibodies in her circulation to cause a transfusion reaction which may be fatal.

Transfusions of Rh positive blood into Rh negative individuals may result also in isoimmunization, and a severe reaction during a subsequent transfusion of Rh positive blood, or as in the example of a mating cited above they may initiate the process of isoimmunization even before the woman becomes pregnant for the first time. It is important, therefore, to test for the Rh in addition to the ABO factors and to use only blood of a suitable type. This is especially important in *pregnancy* and the *puerperium* because of the possibility of the presence of circulating Rh antibodies, and also in all *female patients* from infancy to the end of the childbearing period to prevent Rh isoimmunization by transfusion.

Laboratory Technique.—The simplest and quickest test for the determination of the Rh type is the open slide method. Place on a glass slide a drop of anti-Rh typing serum especially prepared for use in this test. Add a drop of bovine albumin made available in 20% strength. Add two drops of the citrated blood to be tested. Saline suspension of cells may not be used or false negative results will be reported. Mix the typing serum, albumin, and blood together on the slide with a glass rod. Tilt the slide gently from side to side for three minutes, examining it against the background of an opalescent glass plate directly above a lighted 25-watt lamp.

Agglutination is manifested by the appearance of a fine precipitate (agglutinates) resembling red brick dust. It must not be read with the use of the microscope or false positive results may be reported.

Before administering transfusion to Rh negative individuals during pregnancy or in the puerperium it is necessary to do an *Rh cross-match*. A simple and rapid method

is as follows. Place two drops of recipient's serum in a test tube. Add two drops of 20% bovine albumin. Add one drop only of a 2% saline suspension of donor's cells. Incubate the tube containing serum, albumin, and cells in a water bath at 37° for 15 minutes. Centrifuge for 2 minutes at 1000 rpm. Dislodge the cell sediment very gently so as not to break up agglutination which may be present. If no clumping is seen, pour the contents of the tube onto a glass slide and observe under the low power of the microscope for agglutination. If agglutination is present, it indicates that the recipient may be immunized to specific Rh or Hr agglutinogens or other antigens in the donor's cells. Before testing with other donor bloods, the Rh (and ABO) group of the recipient must be carefully rechecked.

Other sources of error in grouping and cross-matching are rouleau formation, cold hemagglutination, bacteriogenic agglutination and isohemolysis. *Rouleau formation* or pseudoagglutination is frequently encountered in conditions of hyperglobulinemia such as multiple myeloma. The serum having this property agglutinates the erythrocytes of any group including those of the blood of the patient from which the serum is derived. *Cold agglutination* occurs in most individuals at low titers. The titer may be increased in certain diseases, especially atypical virus pneumonia. Cold agglutinins are completely inactivated at 37° C. *Bacteriogenic agglutination* or panagglutination may occur with the use of blood contaminated by certain bacteria. The use of fresh or sterile specimens of blood and testing sera will prevent confusion from this source. *Isohemolysis* is caused by the presence in the serum of isohemolysins corresponding to the agglutinins, anti-A and anti-B. By hemolysing the erythrocytes before agglutination is observed, this phenomenon may be a source of error in cross-matching. Such an error may be avoided by the prompt examination of the cross-match.

Therapeutic Aspects.—The treatment of shock with whole blood and plasma as well as the use of plasma in various other conditions and the use of red cell suspensions in chronic anemia have already been described in detail in this and the preceding section.

Transfusion Reactions.—The incidence of transfusion reactions and their occurrence despite the utmost care in the grouping, cross-matching and transfusion of blood afford a constant reminder that transfusion itself may be considered a major surgical procedure and should be undertaken only when definite indications for such therapy exist. The least serious of all is the so-called *allergic reaction*, although usually no allergic basis can be found in either the donor or recipient involved. The usual manifestation is urticaria, and the treatment is symptomatic.

Pyrogenic reactions are the most frequent of all transfusion complications. They are caused by pyrogens which are the polysaccharide fraction of endogenous substances of bacterial lysis present in transfusion equipment which has not been properly cleaned and autoclaved. The use of disposable donor and transfusion sets, guaranteed pyrogen-free by the dispenser tends to reduce the incidence of such reactions. The *symptoms* are chill, increased temperature and pulse, restlessness, dyspnea, and pains in the chest and lumbar region. In a severe pyrogenic reaction there may be circulatory collapse and even death. The *treatment* consists in immediately stopping the transfusion and the application of supportive measures. Since the symptoms just described are also those of an incompatible transfusion, all reactions following the transfusion of whole blood or red cell suspensions should be regarded as being due to incompatibility until proved otherwise.

The *causes* of an incompatible or hemolytic reaction usually are attributable to technical or clerical errors in the laboratory or to carelessness on the part of those responsible

for administering the blood. Because of the possibility of error in grouping or labelling blood, every transfusion of whole blood or red cell suspension should be cross-matched with the patient's blood before being given. This procedure detects such errors and is an additional safeguard in the prevention of reactions. Blood transfusion in many instances is lifesaving, but it can also be fatal. Constant care must be exercised in order to avoid such accidents.

The *symptoms* of an incompatible transfusion are those already described plus the *signs* of hemoglobinemia and hemoglobinuria, followed later by jaundice, oliguria, and in fatal cases, death in uremia. Since the earliest manifestations of a hemolytic transfusion are usually those complained of by the patient, such as a feeling of chilliness, pains in the back, etc., extra care must be taken in giving transfusions to those under general anesthesia or otherwise unconscious. Excluding sudden fatality at the onset of the reaction, renal involvement is the cause of serious consequences.

The *pathogenesis* of the renal damage resulting in the condition known as *lower nephron nephrosis* is not entirely understood. It is doubtful whether hemolysis alone causes damage. Hemoglobinemias and hemoglobinurias are frequently observed in such conditions as the paroxysmal hemoglobinurias without causing renal insufficiency. The various factors involved in the renal damage may be summarized as: lowered blood pressure, vasoconstriction of renal vessels, reduced renal blood flow, and the secretion of large amounts of hemoglobin or its derivatives in an acid urine. *Morphologically* the main alterations in the kidney involve the nephron where degeneration and necrosis of the tubular epithelium occur with heme casts in the distal segments and collecting tubules.

The treatment may be divided into two phases. In the first or immediate phase, on the first indication of a reaction as shown

by the appearance of any of the above-mentioned symptoms, proceed immediately as follows:

1. Stop the transfusion.
2. Obtain a sample of the donor's blood and send it to the laboratory with a specimen of the patient's blood for regrouping and cross-matching and for examination for hemolysis.
3. Give the patient sodium lactate intravenously to render the urine alkaline as soon as possible.
4. Give glucose intravenously to provide fluid for diuresis.
5. Record the 24-hour fluid intake and output, examining the first specimen for hemoglobinuria.
6. Apply heat to the costolumbar regions for reflex relief of renal vasoconstriction.

During the second phase, treatment is directed toward maintaining a balance between fluid intake and output, and preserving the acid base equilibrium until renal function returns to normal. Until such time it is important not to force fluids, since pulmonary edema may develop. The use of the artificial kidney or other dialyzing mechanism should be considered in cases in which there is a gradually rising blood NPN and a decreasing urine output and specific gravity. The products of uremia are thereby removed during the period in which reparative processes may be proceeding in the damaged kidneys.

Other Complications.—A relatively rare complication of transfusion is *virus hepatitis* derived from the donor's blood or plasma. Its incidence is increased by the use of pooled plasma. *Malaria* also derived from the donor may be prevented by excluding donors with a history of malaria. The prevention of the transmission of *syphilis* is discussed later.

Embolism caused by the introduction of air in the transfusion should never occur. The use of specially prepared transfusion sets containing fine mesh filters to remove particulate matter which might be the cause of embolism has already been emphasized.

There is no evidence that, even in massive transfusions of 5 to 10 liters given over a short period of time, the citrate in the transfused blood or plasma has any toxic effect or causes any change in the coagulation time of the recipient's blood. The citrate radicle is rapidly removed from the circulation, oxidized, and excreted.

Pulmonary Edema—The sodium radical may not be excreted rapidly. Its water-retaining properties, especially when large amounts of blood are transfused, may contribute to the development of edema. In such transfusions, furthermore, the amount of sodium citrate may assume significant proportions in patients with cardiac or renal disease. For example, since the ratio of blood to diluent is of the order of three to one, in a transfusion of 4000 c.c. there will be 1000 c.c. of diluent, containing 17 Gm. sodium citrate. It is important, therefore, to use sparingly other intravenous solutions, especially saline (unless specific indications for their use are present), so as to avoid circulatory overloading and pulmonary edema in patients with chronic anemia complicated by cardiovascular or renal disease. In the treatment of pulmonary edema, the performance of a phlebotomy is expedited by the use of blood donor equipment if the donor bottle is of the type which contains a vacuum.

The Blood Bank and Transfusion Services.—The blood bank has become established as an integral part of the hospital. There are, however, other transfusion services organized on a community, or regional basis, and the various Red Cross Societies have developed plans for national blood transfusion services which are in operation in Canada, Great Britain, and the United States. The purpose of both types of services is the same, namely, the provision of adequate amounts of whole blood and plasma properly prepared for immediate transfusion.

It has been conventional to distinguish between direct and indirect methods of trans-

fusion. With *direct transfusion* the donation and transfusion take place simultaneously, requiring that the donor and recipient be side by side. Anticoagulant may or may not be used. The various methods of direct transfusions, now rendered obsolete by the blood bank, are

- 1 Vessel anastomosis

- 2 Vein to vein connection by tubing, valve and pump

- 3 Multiple syringe method.

In the *indirect method*, the proximity of donor and recipient is not required. Donation and transfusion may be separated by minutes, hours, days, or weeks. The use of an anticoagulant is necessary. The procedure may be employed for fresh, stored or preserved blood.

Fresh blood contains the maximum of all components of donor blood and is as effective as in a direct transfusion. *Stored blood*, i.e., blood collected in sodium citrate solution only and refrigerated may be used up to five days following donation, since degenerative changes develop rapidly in the erythrocytes if no preservative is added. When dextrose is added to the blood-citrate mixture to impede the rate of destruction of the erythrocytes, the resultant product is known as *preserved blood*.

Blood is obtained from healthy donors, male and female, between the ages of eighteen to sixty-five. The donors may be:

- 1 Voluntary, i.e., public-spirited individuals

- 2 Friends and relatives of the patient

- 3 Fellow members of a lodge, organization or industry.

Approximately 350 c.c. of blood, under sterile precautions, are withdrawn into a bottle containing a volume of 125 c.c. of a solution of sodium citrate and dextrose. At the same time enough additional blood is taken for filling two tubes, one for serological testing for syphilis, the other to be used for grouping, typing, and cross-matching. The blood is immediately refrigerated. When it

has been grouped for the ABO factors and Rh typed, and if the test for syphilis is negative, the blood is properly labelled and ready for use. Upon receipt of a requisition for blood transfusion, the accompanying sample of patient's blood is grouped and typed. Donor blood corresponding to the recipient's in respect of the factors ABO and Rh is cross-matched with the patient's and if found compatible is marked with the patient's full name, age, sex, ward, and service for proper identification to avoid its being given to the wrong patient. It is then delivered to the ward, operating room, or kept in a reserve compartment of the refrigerator until needed.

All of the above procedures are carried out by the blood bank staff whose grave responsibility in their proper performance is almost unique in medicine. The results of most laboratory tests are used to corroborate a clinical diagnosis, and any discrepancy between the clinical and laboratory findings may be checked by repeating the tests before the treatment is instituted. An error in technique, interpretation or labelling by the blood bank may result in a serious or even fatal transfusion reaction.

The responsibility for the direction of a blood bank may be assigned to an anesthetist, a bacteriologist, pathologist, physician or surgeon. Blood transfusion, because of its many aspects and their rapid development over the past few years, has itself become a specialty. The functions of the director of a transfusion service should include not only the establishment and maintenance of the proper technical and administrative procedures, but also of equal importance, certain other activities. These include the provision of consultative

services in all aspects of blood transfusion therapy, instruction of students and internes in the early recognition of shock, its treatment, and research in the problems related to shock and blood transfusion.

REFERENCES

- Blalock, A., and Mason, M. F.: *Blood and Blood Substitutes in the Treatment and Prevention of Shock, With Particular Reference to Their Uses in Warfare*, Ann. Surg. 113: 657, 1911.
- Cannon, W. B., Fraser, J., and Hooper, A. N.: *Wound Shock and Hemorrhage*, National Research Council Special Report Series 25: 72, 1919.
- Cohn, E. J.: *Blood: A Brief Survey of Its Chemical Components and of Their Natural Functions and Clinical Uses*, Blood 1: 3, 1916.
- Courmand, A., et al.: *Studies of the Circulation in Clinical Shock*, Surgery 13: 964, 1943.
- DrGowin, E. L., Hardin, R. C., and Alsever, B.: *Blood Transfusion*, Philadelphia, 1919, W. B. Saunders Company.
- Denstedt, O. F., Osborne, D. E., Roche, M. N., and Stansfield, H.: *Problems in Preservation of Blood*, Canad. M. A. J. 44: 448, 1941.
- Diamond, L. K., and Denton, R. L.: *Rh Agglutination in Various Media With Particular Reference to Value of Albumin*, J. Lab. & Clin. Med. 30: 821, 1945.
- Kekwick, A., Marriott, H. L., Maycock, W. d'A., and Whitby, L. E. H.: *Diagnosis and Treatment of Secondary Shock, a Study of 24 Cases*, Lancet 1: 99, 1941.
- Keynes, G., ed.: *Blood Transfusion*, Bristol, England, 1949, John Wright & Sons, Ltd.
- Landsteiner, K., and Wiener, A. S.: *An Agglutinable Factor in Human Blood Recognized by Immune Sera for Rhesus Blood*, Proc. Soc. Exper. Biol. & Med. 43: 223, 1940.
- Levine, P., Katzin, E. M., and Burnham, L.: *Isimmunization in Pregnancy, Its Possible Bearing on Etiology of Erythroblastosis Fetalis*, J. A. M. A. 116: 825, 1941.
- Robertson, O. H.: *Transfusion With Preserved Red Blood Cells*, Brit. M. J. 1: 691, 1918.
- Rous, P., and Turner, J. R.: *The Preservation of Living Red Cells in Vitro*, J. Exper. Med. 23: 219, 1916.
- Weil, P. G.: *Shock*, Canad. M. A. J. 46: 307, 417, and 538, 1942.
- Wiggers, Carl, J.: *Physiology of Shock*, New York, 1951, The Commonwealth Fund.

CHAPTER V

PREOPERATIVE AND POSTOPERATIVE CARE

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PREOPERATIVE MANAGEMENT

Assessment of the Surgical Patient as an Operative Risk

Successful surgical treatment means that the surgeon must make not only an exact diagnosis but also carefully plan and execute the operative procedure. This procedure must be carried out without untoward incidents which might lead to the death of the patient or to an unnecessary number of complications. The goal of each hospital admission and surgical procedure must be the early return of maximal possible function of the patient. This end can be gained only by a very careful preoperative appraisal of the patient, not so much in regard to the operative risk, but as to how much the entire phase of hospital treatment will either favorably or adversely affect him.

Patients may be placed into two large categories in respect to their ability to withstand operative procedures:

1. The good risk patient.
2. The poor risk patient.

The good risk patient is one who comes for the treatment of some surgical condition which has caused little or no systemic effect and has no accompanying disease or group of diseases, e.g., uncomplicated hernia.

The poor risk patient is one who presents himself with disordered bodily functions which have been caused by the surgical condition for which he seeks relief, or by other concomitant disease or diseases, e.g., hernia with strangulation where more extensive surgery may be necessary.

It is difficult to categorize each patient. A superficial appearance of reasonably good

health is often misleading and more complete physical or laboratory examination may place this patient in the poor risk category.

Many factors affect the assessment of the patient as an operative risk.

1. **The adjustment of the individual to his disease,** to his attending physician or surgeon, and to the hospital wards and personnel is an important consideration.

2. **Age.**—Infants and young children constitute individual physiological and metabolic problems. They are very prone to develop infection and require special operative techniques. Developmental anomalies which are usually first noticed in this age group will frequently affect the planning and execution of an operative procedure. The elderly patient also constitutes a special problem. He reacts less favorably to the trauma of operation, his nutrition is frequently poor, and he may have accompanying cardiovascular, renal or respiratory disease which necessitates a careful choice of anesthesia and sedation.

3. **Psychological Status.**—It is important that the patient be carefully prepared as to the outcome of any operative procedure. An intimate patient-surgeon relationship is most important in all types of surgery. This is most essential in the handling of a patient with, for example, ulcerative colitis, for whom a permanent ileostomy will be required or one with carcinoma of the rectum who will have a permanent abdominal colostomy.

4. **Degree of Obesity.**—The obese patient is always a poor surgical risk. Technical procedures are much more difficult in such cases, wound healing tends to be delayed,

and the wounds are more prone to become infected.

5. Nutritional Status.—The recognition, prevention and treatment of nutritional deficiencies are important because many surgical conditions may be improved by careful nutritional therapy, and many postoperative complications may be prevented by an adequate preoperative diet.

The assessment of the nutritional status of the surgical patient is made by a careful examination of the patient's dietary history, his weight history, the condition of his tissues, his response to adequate dietary therapy, and by such laboratory aids as estimation of the serum protein levels and the albumin-globulin ratio.

■ Status of the Liver.—The liver is frequently adversely affected by nutritional disorders and dietary deficiencies. All surgical patients must have a sufficient protein and carbohydrate intake because adequate storage of liver protein and glycogen is required to prevent untoward accidents such as the so-called liver failure which occurs sometimes following surgical procedures.

An attempt can be made to evaluate the functional status of the liver by a careful history of diet and weight and by the clinical response to adequate nutritional measures, for example, the disappearance of edema or ascites following high protein intake. In addition the use of various liver function tests can give valuable information. (See section on Liver.)

7. Status of the Kidney.—The function of the kidney must at all times be carefully assessed, as impaired renal function markedly increases the incidence of postoperative complications and possibly may even lead to the death of the patient. An adequate urinary outflow of satisfactory specific gravity is one of the best indications of good renal function.

An estimate of the urinary function may be made by an inquiry into the following factors:

A. The urinary history, e.g., nephritis, prostatic disease, etc.

B. Renal function tests which include:

- (i) urinalysis
- (ii) urine concentration tests, e.g., Mosenthal
- (iii) renal excretion tests, e.g., phenol-sulfonphthalein excretion, and
- (vi) Blood serum estimations: e.g., the serum nonprotein nitrogen.

8. Hematological Status.—The anemic patient does not withstand operation well. He is ill equipped to tolerate operative blood loss and is frequently in a state of malnutrition. It is therefore essential that the hemoglobin and red blood cells be within reasonably normal range prior to surgery, whether the surgical procedure is of an emergency or of an elective nature. The level of the white blood cell count is important as agranulocytosis or leukemia may be present occasionally.

Nutrition

The recognition, prevention and treatment of nutritional deficiencies are important because many surgical conditions may be improved by adequate nutritional therapy, and many postoperative complications may be prevented by careful attention to diet.

The normal individual requires 2,500 to 3,000 calories per day and the diet must include sufficient protein, carbohydrate, fat, vitamins, and minerals. Nutritional deficiencies in surgical patients are relatively common and arise as a result of:

1. Inadequate intake (starvation, ulcer diet, parenteral feedings).
2. Increased requirements (hyperthyroidism, pregnancy, increased muscular activity).
3. Impaired absorption or excess loss (pyloric stenosis, esophageal obstruction, vomiting, diarrhea, intestinal fistula).
4. Defective utilization (diabetes).

Such deficiencies must be corrected before any major surgical procedure is contemplated.

Caloric Requirements.—An adequate caloric intake is necessary to prevent actual or

partial starvation. An inadequate caloric intake results in starvation of the tissues with resultant tissue breakdown and an increase in nitrogen excretion.

The administration of a high caloric diet in a patient with a good appetite can readily be accomplished by an oral intake of high caloric foods supplemented if necessary by high caloric, high protein milk shakes. If the patient's condition is such that oral feeding is impossible or restricted, the problem becomes increasingly difficult. Caloric supplement with isotonic intravenous glucose alone is unsatisfactory as such a large volume of fluids must be given. Intravenous fat, if available, however, offers a means of providing such a high caloric intake without the difficulties which arise when glucose solutions are used.

Tube feeding with carbohydrate, protein, and fat may also be used at times to ensure the necessary number of calories.

Protein.—The body proteins are compounds of amino acids and form a proportion of all living tissues. Ingested proteins are hydrolyzed into their constituent amino acids which are then absorbed from the small intestine.

The plasma proteins consist of three fractions:

1. *Albumin*, which is chiefly responsible for maintaining osmotic pressure.

2. *Globulin*, important in the transportation of immune substances, antibodies and agglutinins.

3. *Fibrinogen*, which is essential for the clotting of blood.

The normal level of the plasma proteins is 7 Gm per 100 ml with an albumin/globulin ratio of 1.5/1. The serum protein, while useful as a guide, is not a true indication of the total body protein, for depleted reserve stores of protein in the tissues may be present with a normal level in the circulating blood. Gross edema usually occurs when the serum albumin is less than 3.0 Gm. per 100 ml. However, the degree of

the edema may be modified by the presence of anemia and by disturbances in electrolyte balance.

There are five main causes of protein deficiency:

1. Insufficient intake of protein to meet the demands of the body; e.g., anorexia, obstruction of the upper alimentary tract and vomiting.

2. Inadequate digestion or absorption of protein; e.g., in chronic gastrointestinal disease, gastrojejunocolic fistula, abnormalities in secretion of the stomach, liver or pancreas.

3. Impaired protein synthesis; e.g., in severe liver disease.

4. Excessive protein loss; e.g., in kidney disease, burns, ascites, ulcerative colitis, or empyema.

5. Increased protein catabolism; e.g., in association with fever, hyperthyroidism, operative procedures, or severe trauma.

The effects of such protein deficiency may cause serious complications in the surgical patient. Every operation increases nitrogen catabolism with a resultant demand on protein reserves, and usually results in a temporary negative nitrogen balance. Protein depletion is increased by hemorrhage, serum loss, and suppuration. Inadequate protein intake is associated with delay in wound healing, wound disruption and chronic indolent ulcers. Hypoproteinemia aggravates the tendency to edema following gastrointestinal operations with resultant obstruction of the anastomotic stoma. The liver, if deficient in protein and especially if low in methionine and cystine, is very susceptible to toxic agents and to the effects of operation.

Correction of Protein Deficiency.—The best way to supply the caloric and protein requirements of a patient is to provide an adequate oral intake. Whole foods, rather than protein hydrolysates, should be used. A diet containing 1 Gm. of protein per kilogram of body weight is adequate for the average pa-

tient, although for short periods this amount may be reduced if sufficient carbohydrate is added. If there has been extensive trauma or severe infection, much larger quantities of protein, up to 300 Gm. per day, may be required. There is evidence that a high protein diet given for a period prior to operation may increase the protein reserves of the body and so compensate for nitrogen loss in the postoperative period.

Protein deficiency in the presence of anemia presents a special problem. If the body is in need of both hemoglobin and plasma protein, the formation of hemoglobin is favored. It is, therefore, most important that existing anemia should be corrected by adequate transfusions of whole blood in protein-deficient patients.

It is essential that the patient be placed in positive nitrogen balance, and that the plasma protein be restored to normal levels before a major surgical procedure is undertaken.

When normal oral feedings are contraindicated, or when only small amounts can be given by this route, other methods must be utilized, e.g., tube feedings with protein hydrolysates or other protein fluids, jejunostomy or gastrostomy feeding or intravenous feeding.

Intravenous administration of whole blood, plasma, serum albumin, and protein hydrolysates can be used in tiding over a patient for a short time when oral intake is impossible or inadequate. Whole blood and blood derivatives are impractical for long-term use. They are expensive, the supply is limited, and the increased blood volume which follows their use may lead to cardiac embarrassment. Protein hydrolysates, reinforced with glucose, can maintain a positive nitrogen balance if given in sufficient amounts. In the usual protein hydrolysate solution there are somewhat less than 50 Gm of protein per 1,000 ml. and there is considerable evidence to show that such hydrolysates are only partially utilized by the body. With the preparations available there

is a high incidence of reactions; nausea, vomiting, fever, and headache. Solutions of pure amino acids are of greater value and are capable of providing adequate protein requirements with minimal or no side effects. However, in the presence of marked protein deficiency, it is impossible to supply sufficient protein by this method.

Vitamins and Minerals.—The administration of additional vitamins and minerals is unnecessary if the patient is taking a full and well-balanced diet. In debilitated patients, and in those for whom parenteral feedings are required, vitamin supplements must be provided. Combustion of carbohydrate greatly increases the requirement for the vitamin B complex and this should be given parenterally to all patients who are receiving intravenous glucose feedings. Vitamin C must also be given as its lack may lead to delay in wound healing. In the jaundiced patient, or in one with advanced liver disease, parenteral vitamin K must be used.

Fluid and Electrolytes.—Seventy per cent of the body weight is composed of water which is contained in two large compartments:

1. Extracellular water 20% (plasma and interstitial fluid).
2. Intracellular water 50% (contained within cell membranes).

TABLE IV
APPROXIMATE AMOUNT AND DIVISION OF BODY FLUID IN A 70 KG MAN

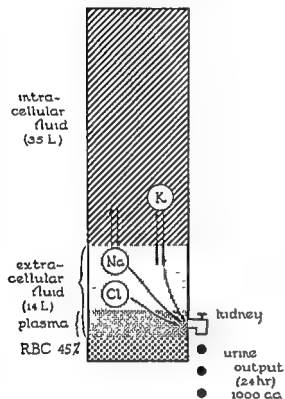
| | % BODY WEIGHT | VOL. IN ML |
|----------------------|---------------|------------|
| I Extracellular | 20 | 14,000 |
| (a) Plasma | 5 | 3,500 |
| (b) Interstitial | 15 | 10,500 |
| II Intracellular | 50 | 35,000 |
| III Total body water | 70 | 49,000 |

Water can move freely between these two compartments in response to physiological requirements. The osmotic pressure in the extracellular fluid is maintained by the sodium ion; in the intracellular fluid by the potassium ion. The distribution of these

two bases is the chief factor controlling the amount of body water in these compartments. Proteins also exert an osmotic effect, but because of their relatively low concentration, as compared to that of the inorganic electrolytes, they have normally little influence on the distribution of the body water.

Protein is mainly concentrated in the plasma and is concerned with the maintenance of fluid within the vascular bed

The normal individual loses approximately 2,300 ml of fluid daily. This elimination occurs in the urine (1,000 ml), in the stool (100 ml), and by vaporization through

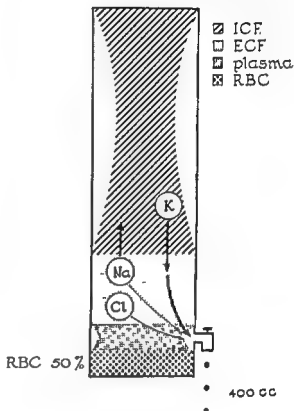


THE NORMAL FLUID SPACES IN A 70 Kg MAN

Fig. 9—A diagrammatic representation of the fluid spaces in a 70 kg man

The volume of the intracellular fluid is shown as 35 liters and that of the extracellular as 14 liters. The concept of continuous shift of Na and K between the intra- and extracellular compartments is indicated by the two directional fine arrows, and that of the continuous K loss by the normal kidney with a normal urine output, by a single fine arrow. There is also a small loss of Na and Cl in the urine due to the kidney mechanisms which preserve the normal body concentration of these ions. The hematocrit is normal and is indicated by RBC 45%.

This series of diagrams depicts the direction of fluid and electrolyte shifts which occur during different degrees and forms of dehydration. The magnitude of these shifts is also roughly indicated.



SLOW OR HYPERTONIC DEHYDRATION

Fig. 10—Slow or hypertonic dehydration

This is a condition which results from water starvation without any extraordinary loss of electrolytes. The dehydration is manifested equally in both the intra- and extracellular compartments. There is increased K loss in the urine as a result of the intracellular fluid shift. There is an increased shift of Na into the cells to compensate for the ionic K loss. There is a slight loss of Na and Cl in the urine as the renal conservation mechanism of these ions has not yet come into effect. The urine output is reduced. The hematocrit tends to be elevated.

Treatment consists of rehydration by isotonic carbohydrate solutions. K may be necessary in late cases.

the skin and lungs (1,200 ml.). The body requirements of water are met by an intake of fluid which comes from two sources: preformed water in fluids and food (2,000 ml.), and water of oxidation (300 ml.). Even when nothing is taken by mouth, the water of oxidation continues to be available to the body.

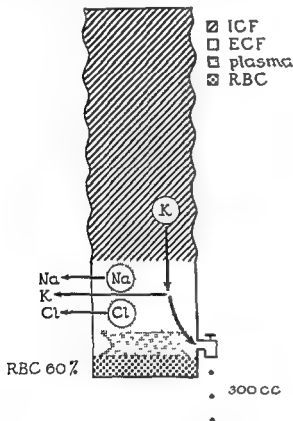
In addition 3 to 5 Gm. of sodium chloride should be given as well as adequate amounts of carbohydrate, fat and protein to satisfy

caloric requirements. An attempt should be made to maintain the urine output at 1,500 ml.

Dehydration

Dehydration results from either a lack of preformed water or an increased excretion of fluid. Water is lost from the body in the following manner.

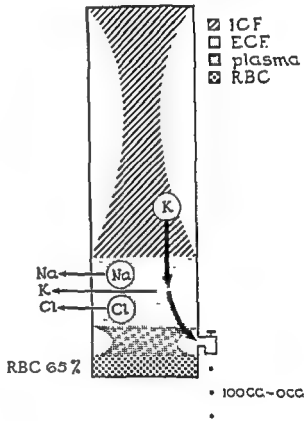
1. Water loss by vaporization which is continuous from the lungs and the skin.



EARLY RAPID OR HYPOTONIC DEHYDRATION

Fig. 11.—*Early rapid or hypotonic dehydration.*

This condition results where there is loss of both water and electrolytes, e.g., in severe vomiting of intestinal obstruction. The magnitude of the electrolyte loss is relatively greater than the fluid loss. The dehydration is initially limited to the extracellular fluid although there is a beginning instability of the intracellular compartment. There is a reduced urine output and a slightly increased K loss. There is no urinary Na or Cl loss, as the kidney is acting to conserve these ions. The hematocrit is significantly elevated.



LATE RAPID OR HYPOTONIC DEHYDRATION

Fig. 12.—*Late rapid or hypotonic dehydration*

This state occurs in untreated severe cases of fluid and electrolyte loss. There is now a marked depletion of water from both compartments. The K loss in the urine is excessive, although the urine output is markedly reduced. There is no urinary Na or Cl loss. The degree of hemoconcentration, as evidenced by the hematocrit, is high.

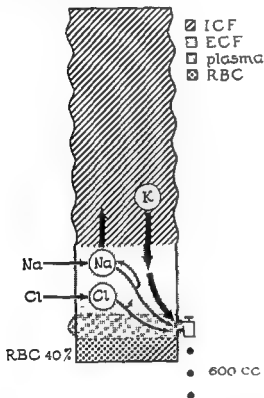
This fluid contains insignificant amounts of electrolytes. If profuse sweating occurs, the water loss is augmented, and there is a loss of larger amounts of electrolytes, chiefly sodium chloride.

2 The daily nitrogenous waste products of the body (35 Gm) are excreted by the kidney. If the kidney is working at maximum efficiency, 500 ml of water are required in the process. If the kidney is

not working at maximum efficiency as is frequently the case in surgical patients, more fluid is required to eliminate these solids.

3 Because the fluid secretions of the gastrointestinal tract are largely reabsorbed, only 100 ml are lost from the body in the stool.

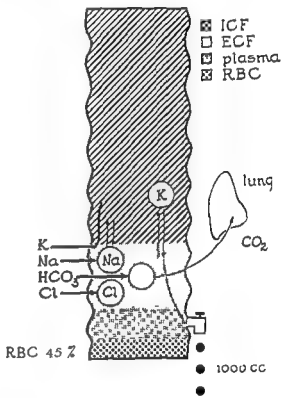
There are two types of dehydration which are commonly encountered in surgical patients



LATE RAPID DEHYDRATION
TREATED WITH NaCl ALONE

Fig 13—Late rapid dehydration treated with NaCl alone

The NaCl solution tends to overhydrate and expand the extracellular fluid compartment and to some extent the intracellular compartment as well. The excess of the Na ion tends to increase Na shift into the cells and to increase K loss in the urine. There is an increased urinary loss of Na and Cl as a result of the kidney's efforts to reduce the overabundance of these two electrolytes. The urine output lags behind the fluid intake. The hematocrit is reduced because of the retained fluid. Clinical edema may be present.



LATE RAPID DEHYDRATION
TREATED WITH BALANCED ELECTROLYTES

Fig 14—Late rapid dehydration treated with balanced electrolytes

The potassium deficit is met by KCl. The Na deficit by NaCl and NaHCO₃. The Cl deficit by the KCl and NaCl. Excess Cl administration is avoided by the use of NaHCO₃, as the HCO₃ is eventually excreted by the lungs as CO₂. The water deficit in both compartments is corrected. The urine output and hematocrit are normal.

1. Dehydration due to *deficient fluid intake* (pure water depletion). This hypertonic dehydration is caused by inability to swallow (dysphagia, coma or great weakness). In these patients the deficiency is of water alone, and the kidney acts to preserve the body stores of extracellular electrolytes. The symptoms are mainly those of thirst and oliguria. The loss is gradual and the extracellular and the intracellular compartments are equally affected. While sodium and chloride are preserved by the selective action of the kidneys, potassium continues to be excreted.

2 Dehydration due to an *increased fluid loss* (hypotonic dehydration). In this condition both water and salt are lost, the electrolyte loss being relatively greater than the fluid loss. This occurs in prolonged gastrointestinal suction, vomiting, diarrhea, gastrointestinal and biliary fistulas. The symptoms are more pronounced and develop more rapidly than in those patients with the hypertonic variety. There is weakness, prostration, hypotension, and hemoconcentration. Thirst and oliguria are not as pronounced as in pure water depletion. The loss in the early stages is mainly from the extracellular fluid compartment with resultant loss of sodium and chloride. It is only in the later stages that any marked depletion of the intracellular compartment occurs with its concomitant potassium loss.

Changes in the acid-base content in the surgical patient are nearly always associated with dehydration and electrolyte derangement. Correction of these factors will restore the acid-base balance.

Treatment

In planning the fluid therapy for any particular patient the type of dehydration must first be recognized and the fluid and electrolyte requirements should be calculated to replace

- (a) the deficit which has already resulted,
- (b) the normal daily requirements, and
- (c) the estimated abnormal loss.

1. *Pure water depletion* may be corrected by oral administration of water alone. If this is impossible or contraindicated, intravenous infusions, hypodermoclyses or intrasternal marrow infusions may be used. The intravenous route is preferable, except in infants where hypodermoclysis may occasionally be indicated. Intravenous fluids should be made isotonic by the addition of 5 % glucose or invert sugar. Carbohydrate has the added feature of reducing the catabolism of protein. The kidneys therefore have less nitrogenous waste products to excrete and also the oxidation of the carbohydrate itself produces a small amount of water. In severe cases of simple water starvation, potassium must also be given.

2. *Water and electrolyte depletion.* These patients must be treated by the administration of water plus inorganic electrolytes. Large amounts of fluid and salt are necessary to correct the deficiency in the severely dehydrated patient. If the serum sodium and chloride are markedly depleted, hypertonic saline solutions must be used. A potassium deficiency frequently accompanies this type of dehydration and must be corrected.

POSTOPERATIVE MANAGEMENT

Pain

Every surgical operation is followed by a period in which the patient needs special care in order to minimize his discomfort and to prevent the development of complications. Pain in the operative area is often accentuated by the anxiety of the patient. The most effective method to relieve pain is the hypodermic injection of morphine sulphate, gr. $\frac{1}{8}$ - $\frac{1}{4}$ (7.5 - 15 mg.), repeated at four-hour intervals when necessary. This drug should be used with caution, especially in the older age groups because of its depressant effect on the respiratory center. Demerol, 100-150 mg, may be substituted for morphine. This drug produces less respiratory depression and does not cause the unpleasant side effects of

nausea and vomiting which so frequently occur following the administration of morphine. It should only be necessary to continue these drugs for the first 24 to 48 hours postoperatively. If discomfort persists, milder analgesics such as codeine and acetylsalicylic acid may be given. Apprehension is best relieved by reassurance, supplemented in some cases by suitable barbiturates.

Fluids

In the immediate postoperative period, the fluid requirements of the surgical patient are increased due to operative blood loss, increased sweating, gastric or other intestinal drainage and vomiting. In the absence of nausea and vomiting, the oral administration of water may be started in small amounts as soon as the patient has recovered from the anesthetic and increased as rapidly as his tolerance permits. If the patient cannot take his fluid requirements by mouth, parenteral fluids should be given in such an amount as to ensure a urinary output of 1,000 to 1,500 ml daily.

Diet

The details of diet vary widely with various operative procedures. The aim should be to return the patient to a full and adequate oral intake as soon as possible. Following minor surgical procedures, and in the uncomplicated case, the patient can take enough food to maintain his nutrition, but after major surgical procedures, it is frequently necessary to supplement the oral intake by parenteral means.

Care of the Lower Gastrointestinal Tract

The diet during the first few postoperative days ensures little residue. There is, therefore, no necessity for an evacuation before the third postoperative day. During this time, a rectal tube may be used to relieve flatus. If the bowels do not move by the third or fourth postoperative day, a small

enema may be given. If enemas are ineffectual, rectal examination should always be done to exclude impaction of feces in the rectum. Fecal impaction can also produce symptoms of diarrhea and tenesmus.

Ambulation

The patient should be turned on alternate sides every hour and should be encouraged to breathe deeply and to cough. His respiratory excursions may be increased by the administration of carbon dioxide (5%) and oxygen. The patient should be allowed out of bed as soon as possible, but each patient must be judged individually. His general condition, the nature and extent of the operation, the presence or absence of complications must be considered. It is important to insist on early active movement. The practice of lifting a patient into a chair where he remains immobile until lifted back into bed does not constitute early ambulation. Many patients including those who have had abdominal operations may be allowed up in 24 hours, allowed to walk to the bathroom after 48 hours, and within 4 to 5 days may be up and about most of the time.

Such a program adds immeasurably to the well being of the patient and results in fewer pulmonary complications, briefer convalescence, and easier nursing care. The incidence of wound disruption and postoperative hernia have not been increased. However, phlebotrombosis and subsequent pulmonary embolism may still occasionally occur in patients who have followed this regime.

Care of the Wound

In the so-called clean case, in the absence of fever, undue pain, or discharge in the operative area, the dressings should not be disturbed until the time for removal of sutures. Heavy dressings should be avoided. Repeated inspections of the wound are unnecessary, add to the patient's discomfort, and may predispose to infection.

No set rules can be given as to the time of removal of sutures. Sutures on the face and neck should be removed early (second to fourth postoperative day). In other situations (e.g., abdomen) superficial skin sutures are usually removed on the sixth to seventh postoperative day, while deep retention sutures should remain in place for 12 to 14 days. Michel clips should be removed from abdominal wounds on the fourth to fifth day, and from neck wounds on the second to third day.

If a drain has been used, a thick absorbent dressing is required to absorb the discharge; this must be changed when necessary. No rules can be given for the removal of drains; the proper time for removal will vary with the purpose for which they were inserted. If the drain has been used to guard against the collection of serum or blood, it may be removed in 24 to 48 hours. When drains are used as a protection against leakage (e.g., from a cystic duct or gastrointestinal anastomoses), they should remain in place until the possibility of such a leak has passed.

Complications

Respiratory Tract Complications.—*Atelectasis* is the commonest pulmonary complication and its incidence is about equal following general or spinal anesthesia. It is caused by the accumulation of bronchial secretions which occlude the main bronchus or one of its branches. Subsequent absorption of air distal to the obstructing plug results in collapse of the lung parenchyma. If the obstruction is not relieved, secondary infection occurs with the development of a lobular pneumonia.

Clinically, atelectasis should always be suspected in the presence of a sharp rise in temperature in the first 48 hours following operation. If large segments are involved this may be accompanied by an increase in respiratory rate and cyanosis. The diagnosis can be made readily by physical signs. The mediastinum is shifted to the side of the lesion and the diaphragm on the in-

volved side is elevated. The area is dull to percussion and breath sounds are decreased or absent; râles are commonly present. The diagnosis may be confirmed by roentgenological examination.

The treatment is chiefly prophylactic. Oversedation in the preoperative and postoperative periods should be avoided and the bronchial tree must be kept free from secretions by hyperventilation, enforced coughing, and tracheal aspiration.

In the established case the plug must be removed and the lung re-expanded. This can usually be accomplished by enforced coughing and tracheal aspiration. If the lung does not expand rapidly, bronchoscopic drainage should be done early and repeated as often as required. Antibiotic therapy should be given to aid in the control of secondary infection.

Pulmonary Embolism.—Pulmonary embolism usually follows thrombosis of the veins of the legs and pelvis. In contrast to pneumonia and atelectasis, pulmonary embolus rarely occurs before the end of the first week after operation. Fatal embolism is often preceded by smaller, nonfatal infarcts.

The clinical manifestations depend upon the size of the embolus and its location. If a main pulmonary artery is occluded, the symptoms are those of acute respiratory distress with circulatory collapse. If a smaller branch is blocked, the patient has pain in the affected side of the chest, dyspnea, cyanosis, fever and hemoptysis. X-rays in the early stages, frequently fail to demonstrate the lesion.

Treatment.—Careful daily examination of the legs for signs of thrombosis should always be done. At the first sign of involvement of the veins, anticoagulant therapy or venous ligation should be carried out. If pulmonary embolism occurs, antishock measures should be instituted and antispasmodics such as papaverine should be given. Oxygen and anticoagulant therapy must be continued.

Fat embolism usually follows severe trauma and fractures and is due to the presence of fat globules in the circulating blood.

The symptoms are predominantly cerebral or pulmonary. The patient is restless, confused, and possibly comatose and may develop dyspnea, cyanosis, and signs of pneumonia. Petechiae are often present over the upper thorax and neck. The diagnosis is established by the presence of fat in the sputum or urine. Treatment is directed toward the relief of shock and pulmonary complications.

Gastrointestinal Complications

Nausea and Vomiting.—Nausea and vomiting are common in any patient who has had a general anesthetic and are aggravated by excess intake of fluids or food in the immediate postoperative period. Many patients are sensitive to morphine which may prolong and increase vomiting. If vomiting is prolonged, it becomes potentially dangerous because of the resulting dehydration and chloride loss. In such cases, gastric lavage, or continuous gastric suction should be instituted and parenteral fluids given.

Distention.—Following every abdominal operation a certain degree of temporary paresis of the intestines occurs, but the bowel musculature quickly regains its tone. Accumulation of gas within the intestinal lumen, and the ineffectual efforts to expel it, result in the so-called *gas pains*. The atony persists in some cases, due to prolonged or rough handling of the bowel, retroperitoneal hemorrhage, peritonitis or operations on the retroperitoneal tissues. If this complication is not recognized early and treated promptly, increasing distention will result. The major amount of intestinal gas is derived from swallowed air with only a small proportion coming from fermentation of food and interchange of gases with the blood stream. It is much easier to prevent than to treat *ileus*. In the early case, a rectal tube may permit the passage of flatus with marked relief. Early prompt use of gastric in-

tubation with continuous suction will prevent distention in the majority of cases. Intestinal intubation with the Miller-Abbott, Harris or Cantor tubes will overcome ileus if the tubes can be passed into the small bowel. Approximately 70% of the intestinal gas is nitrogen and routine inhalation of a high concentration of oxygen may assist the body in eliminating excess nitrogen. Drugs have a limited field in the treatment of intestinal ileus. Morphine enhances both the tone and the peristaltic action of the intestine, especially of the small bowel. Prostigmine, which acts chiefly on the small intestine, and Pitressin, which stimulates the large bowel, may be useful. (See section on Intestinal Obstruction.) However, as a rule no drugs appear to be of value until peristalsis is resumed.

Acute Dilatation of the Stomach may follow any operation and is regularly associated with paralytic ileus. There is loss of gastric tone, and large quantities of gas and fluid accumulate in the stomach. The enlarged dilated stomach can be palpated, and large quantities of fluid are vomited. The treatment depends upon early diagnosis and the immediate removal of the contents of the dilated stomach. Continuous suction on the stomach tube should be maintained until the gastric tone is restored.

Hiccough.—Postoperative hiccough may occur following operations on the abdominal viscera, the genitourinary tract, the diaphragm, or the central nervous system. It is thought to be a reflex phenomenon due to stimulation of the afferent nerve terminals in the diaphragm. It is usually transitory and responds to symptomatic treatment, but it may be prolonged and very refractory to therapy.

Inhalation of carbon dioxide (5 to 10 % in oxygen) is often effective. As hiccough frequently accompanies an ileus or stenosis in the intestinal tract, gastric suction may bring relief. In the intractable case it is occasionally necessary to interrupt the phrenic nerve, either by local anesthesia or

operative procedures. If this is done the patient should be first fluoroscoped in order to determine which side of the diaphragm is producing the hiccough.

Parotitis is an uncommon postoperative complication. The invading organism, usually a staphylococcus, enters via the parotid duct. A dirty mouth in a dehydrated patient is undoubtedly a predisposing factor and the condition generally occurs in debilitated individuals.

Pain, swelling, and tenderness in the parotid region accompanied by pyrexia appear within a few days of operation and pus can often be expressed from the opening of the parotid duct.

The treatment of oral sepsis, and the prevention of dehydration will prevent the occurrence of parotitis. Once the infection is established, antibiotic therapy is of great value combined at times with small doses of x-ray to the parotid region.

Urinary Complications

Urinary Retention.—Inability to void is common in the immediate postoperative period and is, as a rule, transitory. Many patients experience difficulty in voiding while recumbent, getting the patient out of bed frequently enables him to empty his bladder. Parasympathomimetic drugs are sometimes useful. If such measures fail, catheterization is necessary. Overdistention of the bladder predisposes to infection, and catheterization should be done before this occurs. Great care must be taken to avoid introducing bacteria into the bladder during this procedure. If retention persists, an indwelling catheter may be introduced, and tidal drainage is occasionally indicated.

Renal Failure may complicate surgical operations especially if they are accompanied by hypotension and shock. Dehydration and previous kidney disease are predisposing factors. Following severe burns, incompatible blood transfusions, etc., lesions of the lower nephron nephrosis type occur and the func-

tional derangement may lead to complete anuria. In many cases the kidneys begin to function spontaneously after a few days. Great care must be taken that the patients are not overloaded with fluids during this period.

Wound Complications

Infection should not occur in a *clean* case. If it does, a careful review of the operation and dressing techniques must be made. Infection is usually restricted to a small part of the wound, but a generalized wound infection and spreading cellulitis may occur. Clinically the patient runs a normal course for several days and then develops fever and pain at the operative site. Examination of the wound may show swelling and redness, but often a localized point of tenderness is the only indication of underlying infection. The wound should be reopened, the pus evacuated, and antibiotics administered.

Hematoma may occur in any operative wound. Its incidence is inversely proportional to the surgeon's care in effecting hemostasis. Small hematomas will be absorbed spontaneously but predispose to infection. Large hematomas should be evacuated under anesthesia and the wound resutured, as retained blood clot markedly delays wound healing.

Disruption of the wound occurs more commonly in the aged and obese, in patients with debilitating disease such as cancer and in those who experience vomiting, distention or cough in the postoperative period. Incomplete wound disruption is the basis for almost all incisional hernias. It commonly occurs about a week after operation.

The patient's nutritional status should be satisfactory before operation. The incision must be closed carefully and without tension on the suture line. The wounds of debilitated patients should be closed with through-and-through nonabsorbable sutures.

Treatment.—The wound should be immediately resutured unless the patient's condi-

tion is so critical that any operation is contraindicated, in which event adhesive strapping may serve as a temporary measure.

PATIENTS PRESENTING SPECIAL PROBLEMS

Infants and children are particularly susceptible to disturbances in metabolism as a result of operative trauma. They require a much greater caloric, water and salt intake per kilogram of body weight than do adults.

The effects of dehydration are more profound, and occur more rapidly in infants and young children. (See pyloric stenosis.)

The elderly patient reacts less favorably to trauma than does a younger person. Advanced age is frequently associated with nutritional deficiencies, cardiovascular, renal or respiratory disease, any of which will add to the operative risk. However, with careful preoperative and postoperative care, proper choice of anesthetic agents, and meticulous surgical technique, the average elderly patient will come through even an extensive surgical procedure without difficulty.

The postoperative phase of treatment is most important, for patients even with outwardly good cardiovascular or renal function may develop a breakdown of these systems, and special precautions must be taken to forestall any such event. An adequate urine output is most important to ensure that the nitrogenous waste products are excreted, because the renal concentrating power may be reduced.

To help guard against cardiac complications, salt intake should be restricted for several days prior to any major surgical procedure. Intravenous feeding must be done with great care to avoid overloading the circulation with ill-chosen or rapidly administered solutions.

Respiratory complications frequently develop in the elderly patient from concomitant chronic respiratory disease or from inability to cough up retained bronchial secre-

tions. Assiduous nursing care and prophylactic chemotherapy will prevent most complications of this type.

The Patient With Cardiac Disease

The patient with cardiovascular disease, whether young or old, who undergoes a surgical procedure does so with added risk. However, except in the presence of cardiac decompensation, or following a recent cardiac accident, or in severe congenital heart disease, this risk should not be inordinately great.

Patients with chronic rheumatic heart disease, angina pectoris, and hypertension withstand extensive surgical procedures without difficulty if certain precautions are observed. The preoperative sedation should be sufficient to allay apprehension, yet not enough to cause marked cerebral or respiratory depression. Anesthetic agents must be administered with extreme care to avoid struggling during the stage of excitement and anoxemia and hypotension must be prevented at all times. Intravenous fluids must be given very carefully to avoid overloading the right side of the heart, with the dangerous consequence of pulmonary edema. Non-electrolyte crystalline solutions are usually well tolerated, but sodium chloride is best avoided because of its tendency to increase the blood volume. Whole blood and blood plasma should only be given to replace their loss.

The most important practical point in assessing the operative risk in cardiac patients, is the degree of exercise tolerance and the presence or absence of cardiac decompensation. The use of digitalis and quinidine is not indicated unless cardiac irregularity or failure develops during the postoperative period.

The Patient With Renal Disease

The patient with chronic renal insufficiency constitutes a great problem to the surgeon. The impaired powers of concen-

tration and selective absorption of the renal tubules require that special attention be paid to fluid and electrolyte requirements to ensure adequate excretion of waste products and a satisfactory electrolyte balance. The greater the degree of impairment of function, the greater the urine volume required. Renal failure or shutdown can occur with great suddenness in cases with impaired renal function. This usually occurs after a period of hypotension or dehydration and these events should be avoided at all costs. Acute renal disease such as pyelitis and nephritis should be treated before any but emergency surgical procedures are carried out.

The Patient With Respiratory Disease

Chronic respiratory disease augments the risk by leading to an increased incidence of postoperative pulmonary complications. With the impaired respiratory excursion and the deficient aeration of the lungs most anesthetic agents are poorly tolerated. Careful attention to oxygen supply and a free airway are most essential during operations on patients with chronic respiratory disease. Postural drainage with parenteral and aerosol chemotherapy should be used during the preoperative and postoperative phases.

Acute respiratory infections necessitate the postponing of elective surgical procedures. If an emergency condition arises, inhalation and parenteral chemotherapy should be employed. In any case of pulmonary disease, particular effort must be directed to the clearing of tracheal and bronchial secretions by coughing, tracheal aspiration, early movement, and ambulation.

The Obese Patient

Obesity, particularly in the older patient, increases the operative risk, first, because of its frequent association with metabolic disturbances or with organic cardiac or respiratory lesions, and second, because of

the difficulties encountered in carrying out surgical procedures, especially those within the abdomen.

Weight reduction should be attempted prior to elective surgical procedures. However, in the event of emergency, the added risk must be accepted.

Diabetes in the Surgical Patient

Surgery in the case which is unrelated to the diabetes. The surgical management of the diabetic patient necessitates close cooperation between physician and surgeon. If the hyperglycemia is not controlled, the incidence and severity of infections will be increased, delayed wound healing may occur, and glycosuria, acidosis, and coma may result. In the presence of diabetes, operations may be performed with comparative safety so long as the diabetes is under control. It should be remembered, however, that the operative trauma will temporarily aggravate the diabetic state.

Emergency operations present a difficult problem. In infections and trauma, the needs of the body for insulin are increased and the dosage must be adjusted accordingly. The operative risk in these patients is greatly increased. If a true emergency exists, every attempt should be made to control acidosis, but if the added risk is both necessary and appreciated, it may still be advisable to proceed with surgery even in the presence of ketosis.

The acute abdominal case is of special interest. In many of these, e.g., acute appendicitis, the symptoms are milder in the diabetic. It should also be appreciated that in patients in diabetic acidosis or impending coma, acute abdominal symptoms may develop which may be easily confused with those of appendicitis and pancreatitis.

Localized collections of pus in the diabetic patient may be called a diabetic emergency. The presence of retained pus has an adverse effect upon the diabetes, making its control

difficult or impossible. As drainage operations, outside the abdominal cavity, are of relatively short duration and can be done under light anesthesia, operation can be undertaken without delay even in the presence of uncontrolled diabetes.

Preoperative Care.—It is important that the glycogen and protein reserves of the liver of the diabetic patient be augmented by a diet containing adequate amounts of carbohydrate and protein. Because of the widespread arteriosclerosis, a careful evaluation of the cardiac and renal status should be made before any elective operation.

In general, protamine insulin is more difficult to use in the immediate preoperative and postoperative periods, and so a change should be made to the quick acting crystalline form which permits more exact regulation. On the day of operation the caloric needs of the patient can be met by intravenous glucose which should be covered by insulin (1 unit of insulin per 5 Gm of glucose). No attempt should be made to render the urine completely sugar free because of the dangers of hypoglycemic reactions. It is safer to maintain a moderate degree of hyperglycemia in the preoperative and postoperative periods than to risk the development of insulin shock.

Anesthetic.—In the selection of an anesthetic, three factors should be kept in mind.

1 *Toxicity* General anesthetics, especially chloroform and ether, should be avoided if at all possible because there is a risk of producing fatal damage to a glycogen depleted liver.

2 *Length of action* The sooner after operation the patients are able to take carbohydrate food, the easier will be the management and the less likely the development of ketosis.

3 *Liability to produce postoperative vomiting.* The anesthetic of choice, therefore, is one with a low toxicity, rapid elimination, and a low incidence of nausea and vomiting. For operations of a minor nature, nitrous

oxide and oxygen should be used. Even in abdominal surgery these gases combined with curare, seem free from deleterious effects. For major surgery, both nitrous oxide supplemented with curare and spinal anesthesia are the agents of choice.

Postoperative Care.—An adequate fluid intake is important in the diabetic patient. During the operative period blood and saline may be used as necessary. Glucose, if required in the postoperative period to meet caloric requirements, should be covered by insulin, though care should be taken to avoid hypoglycemia. Oral feedings should be given as soon as tolerated and increased until an adequate diet is attained.

Surgical Disease Occurring as a Result of Diabetes.—The surgical diseases which occur as a result of diabetes are the result of sepsis and ischemia. The diabetic patient has a lowered local and general resistance to infection on account of cardiovascular disturbances (primarily arteriosclerosis) and abnormal carbohydrate metabolism. Fully developed sepsis is resisted very poorly by the diabetic, and treatment should be directed toward the infection rather than the diabetes.

While infections in the diabetic patient can occur anywhere, those of the feet deserve special attention, not only because they form the largest group, but also because their treatment is often complicated by the presence of ischemia.

Surgical conditions of the feet in diabetics must be considered under three headings.

1. Infection without ischemia.
2. Infection with ischemia
3. Gangrene

INFECTION WITHOUT ISCHEMIA

A carelessly cut toe nail or corn is often the starting point of an acute infection. This may remain localized and require only minor surgery or may spread rapidly so as to endanger the limb and even the life of the patient. Because of the rapidity of spread and

the increased severity of the diabetes in the presence of infection, these conditions must receive prompt attention. Clinical manifestations of inflammation (pain, heat, redness, and tenderness) are not so marked in the diabetic and the differentiation between cellulitis and an abscess cavity may be very difficult for this reason. Moreover, the infection spreads so readily along fascial planes and tendons that pus may not be under sufficient tension to produce fluctuation. Infection of bone with resultant osteomyelitis may occur even when the infection is uncomplicated by ischemia. The involved part should always be x-rayed in order to exclude this complication.

Treatment.—In the absence of ischemia, the treatment of infection need be no different than in the nondiabetic patient.

1. The infected part should be put at rest.
2. Adequate chemotherapy should be given.
3. Incision with drainage is indicated when pus is present. Infections are more common in the foot, but they may occur anywhere, and the spread is equally rapid, making early and adequate drainage a necessity.

INFECTION WITH ISCHEMIA

This is the commonest and the most serious surgical complication of diabetes. The arteries of the upper extremity are rarely involved by arteriosclerosis obliterans and if one artery is so affected, the collateral circulation is usually sufficient to prevent distal ischemic changes. It is the lower extremities which invariably are involved in this disease.

The ischemia is marked in all parts of the foot, and trophic changes are common. The skin is thin and shiny, and has lost its elasticity. The nails are frequently thickened and deformed. The skin over the sole is dry and fissured, providing an easy entrance for bacteria.

If infection occurs in such a limb the results vary with the degree of the ischemia.

Treatment.—There are three courses of action which can be followed in such a case:

1. *One-stage operation after the infection has been controlled* and the amount of damage to underlying bone assessed. The majority of cases fall into this group. The operation is planned according to the clinical and radiographic findings and is carried out when the patient's diabetic condition is fully controlled. The objects of the operation should be to establish free drainage, remove all dead and infected bone, and leave the patient with a useful foot. Extensive incisions should be used.

2. *Preliminary drainage* to relieve pain and improve the general and diabetic condition with a view to further local surgery after the acute phase has passed. Simple drainage should be combined with chemotherapeutic coverage.

3. *Amputation of the limb without trying to preserve the foot.* This is at times a life-saving measure but is less often necessary since the advent of modern chemotherapy.

When the infected area has been laid open, local irrigations may help to clear up the infection, although care must be taken to avoid maceration of the tissues. Dry heat must never be used in an ischemic foot because of the danger of producing a burn, the results of which may be disastrous.

GANGRENE

Lower extremity gangrene occurs in 5% of diabetic patients. It does not seem to be related to the severity of the diabetic condition but is directly proportional to the degree of vascular occlusion.

DRY GANGRENE

Localized dry gangrene of one or more toes is relatively common in the elderly diabetic even when the diabetes is well controlled. This condition should be treated as conservatively as possible.

Treatment.—Prevention is easier than the actual treatment of the established case.

Unnecessary amputations may result from the neglect of apparently trivial injuries of the feet. Epidermophytosis is a particular danger as it predisposes to secondary infection

Conservative treatment is frequently possible provided meticulous care is given to the local condition. Bed rest is imperative at the first sign of impending gangrene. The object of local treatment must be to prevent the development of secondary infection in the gangrenous area, and every precaution must be taken in dressing and handling the part. The affected toe or toes should be covered with sterile dry dressings and heat should not be applied.

The best results are obtained by allowing the part to separate spontaneously, but amputation must often be considered. The surgeon, in deciding upon the level of the am-

putation, must consider both the basic pathological process and the level of effective collateral circulation. (See *Peripheral Vascular Disease*.)

INFECTED OR WET GANGRENE

The treatment of gangrene when infection has set in is essentially the same as that described under *Infection With Ischemia*. In these cases, however, amputation is more often necessary.

REFERENCES

- and Pathology of an Extracellular Fluid: a Lecture Syllabus, Cambridge, Mass, 1947, Harvard University Press
Mason, R. L., and Zintel, H. A.: *Preoperative and Postoperative Treatment*, ed 2, Philadelphia, 1946, W B Saunders Company

CHAPTER VI

INJURIES DUE TO PHYSICAL AGENTS

THERMAL, IRRADIATION, ELECTRICAL AND CHEMICAL TRAUMA

HAMILTON A. BAXTER, M.D.

THERMAL BURNS

The prevalence of injuries from heat may be realized when it is shown that nearly 8,000 people in the United States, and an estimated 60,000 in the world die each year from this cause. These figures become insignificant when compared with the hundreds of thousands of people who are hospitalized for this type of injury and suffer various degrees of disfigurement each year.

Between World Wars I and II certain marked physiological disturbances in the burned patient were observed. First the deficiency of electrolytes and fluid was noted. Then it was shown that plasma was extravasated into the burned area with resultant hemoconcentration and that plasma administered intravenously was beneficial. Finally the infective phase of burns was recognized and various therapeutic measures advocated for treatment. As a result of the large number of burns suffered by civilians and military personnel in World War II, intensive research was carried out, and several major advances in therapy resulted. These may be summarized as follows:

1 The development of improved methods of treating shock and hemoconcentration.

2 The wide adoption of the pressure dressings of Allen and Koch.

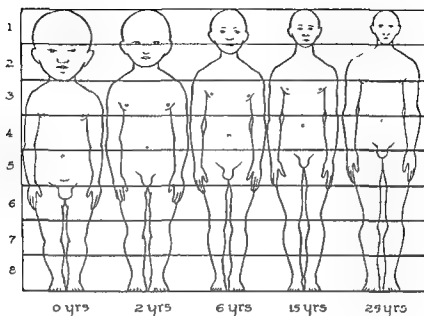
3 The dermatome, which enabled surgeons to obtain accurately and easily any thickness and amount of skin grafts desired.

4 The discovery and use of sulfonamides, penicillin, and newer broad spectrum antibiotics

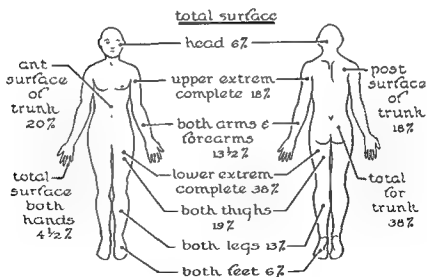
5. The resurfacing of granulating areas at the earliest possible time, which automatically prevents most of the chronic sequelae of burns.

The original classification by Dupuytren of the severity of a burn into six degrees was simplified for reasons of practical convenience to include only the following three divisions: First degree burn, which causes reddening of the skin; second degree burn, which involves the deeper layers, but regeneration is possible from the remaining hair follicles, sweat and sebaceous gland ducts; and third degree burn which destroys the entire skin and any or all deeper structures, including bone. Thus, in modern terminology, the third degree burn includes the fourth, fifth and sixth classifications by Dupuytren. Healing of a first degree burn is complete in a few days, while one of second degree will require about two or three weeks depending on the number and viability of epithelial structures remaining. A variable amount of scar formation results. The third degree burn, if small, may heal by epithelial ingrowth from the edges or contracture, but large areas should be skin grafted as soon as the depth of the burn is evident. Although a number of methods for determining the depth of thermal burns have been proposed, the most reliable test is that of visual inspection at intervals, due allowance being made for the region of the body and the usual hair distribution.

The Berkow table for adults and its modification for children should be used to calculate the percentage of the body surface



A



B

Fig 15—A A comparison is made of the changes in per cent surface area of the head and extremities between children and adults

B. Berkow's method of calculating the extent of burns

burned so that the amount of plasma and fluids to be administered may be determined. A rough estimate of the areas of first, second, and third degree burns should be charted for comparison with the final outcome.

Physiologic Derangements

Shock.—Immediately following a burn, pain is experienced which varies with the type of injury. This may cause syncope or death within an hour or two despite prompt therapy.

Blood Flow.—Due to the painful sensations there is a brief period of blanching of the skin followed by vasodilatation of the vessels of the skin and subcutaneous tissue. Unless charring has occurred there is a marked increase in capillary permeability with extravasation of plasma, particularly the albumins, into the interstitial spaces. The more extensive the burned area, the greater will be the amount of plasma lost from the vascular system. This results in hemoconcentration, which is well established in 24 hours and which is so characteristic of thermal burns.

Loss of Plasma and Increase in Lymph Flow.—The protein content of the escaped fluid is estimated at about 4 % and from 0.4 to 2.3 grams of nonprotein nitrogen may be lost per square inch of burned surface in 24 hours. The flow decreases as the burn heals. The lymph return is increased from a burned area, and this flow is not reduced by an occlusive burn dressing. No definite toxic substance has been found in the lymph returning from an area of thermal burn in man. However, it has been shown that transfusion of blood from a severely burned dog will cause cessation of secretion in the isolated, denervated stomach of another dog and that a hemolysin is present in lymph from burned areas.

Anemia.—Immediately following the burn there is local heat destruction of red cells in the area of burned skin and subcutaneous

tissue. This anemia is temporarily masked by the hemoconcentration which occurs. Due to hemolysis, dilatation of capillaries and stasis, the circulating mass of red blood cells per unit volume of uninjured tissue may be reduced by as much as 50 %.

Permeability.—The burned surface permits flow of diffusible substances in either direction. Sulfonamides given orally appear in the exudate and if applied locally may be absorbed into the blood stream in dangerously large amounts. Penicillin, aureomycin and other antibiotics are present in the plasma exuding from the burned surface in amounts which would encourage bacteriostasis. Although bacteria do not invade intact epithelium of a burned surface even if coagulated, once this defense is broken, they may cause local inflammation or bacteremia if favorable conditions arise.

Systemic Manifestations

Primary shock is of neurogenic origin and results in generalized vasomotor collapse. It is seen shortly after injury and is characterized by pale, clammy, moist skin, shallow respirations and weak pulse. In severely burned patients death may occur from this cause before a physician can institute treatment, or even in spite of it.

Secondary shock occurs subsequent to primary shock, lasts for two or three days, and is characterized by hemoconcentration and the escape of plasma into the tissues.

Due to the increased red blood cell concentration and increased viscosity of the blood there is decreased cardiac output and decreased blood flow to certain vital organs. The liver and kidney are particularly affected and due to decreased blood flow, renal function may be drastically reduced so that the tissues and tubules of the kidney may never regain their function, and death from uremia may result.

The normal functions of the liver are retarded when there is an inadequate supply of

blood and oxygen, so that glycogen cannot be stored, and deamination is prevented

Lungs.—Direct exposure to flame or hot gases may cause some injury to the respiratory tract, usually only as far as the trachea. The bronchiolar epithelium is more likely to be injured by chemically irritating gases. Carbon monoxide poisoning and lowered oxygen content of air may become a major lethal factor when fire occurs in enclosed spaces

conditions the adrenal may not be able to react adequately, with subsequent collapse.

Acute toxemia overlaps secondary shock and lasts for seven to ten days. Those who survive secondary shock and subsequently die, usually do so in this period. Whether it is due to toxic substances, physical changes in the circulating blood, or is caused by anemia and septic infection is uncertain. However, general use of the newer antibiotics has reduced the systemic and local



Fig 16—Marked emaciation which develops in patients with extensive third degree burns when skin grafting is delayed beyond the optimum period

Adrenals.—Following burns as well as other forms of stress there is adrenal hyperactivity mediated through the pituitary with increased output of adrenal hormones as revealed by eosinopenia and a rise in output of urinary ketosteroids. In certain systemic

effects of pathogenic organisms to a minor role, while early and frequent blood transfusions eliminate the red cell deficit. Nevertheless, in some patients the pulse becomes rapid, the skin cold and cyanotic, and hyperpyrexia may develop. Restlessness progress-

ing to stupor and a final fall in blood pressure indicate approaching death. In children, convulsions often precede the demise. Sudden respiratory failure may occur and increased intracranial pressure with compression of the medulla oblongata has been found. Microscopically, severe interstitial edema and ganglion cell changes most marked in the hypothalamus have been noted.

Chronic sepsis is due to the retention of pus under dead skin or wound granulations, and is eliminated by removal of eschar and skin grafting.

Treatment

General.—

1. Give morphia, gr. $\frac{1}{4}$. Wrap patient in clean linen or blankets and transport to hospital.

2. According to Harkins, shock may be treated by giving undiluted plasma and electrolyte solution on the basis of 100 c.c. for every point the hematocrit exceeds 45 in a person weighing 150 pounds. Isotonic electrolyte solution made up, two-thirds of isotonic sodium chloride and one-third of $\frac{1}{6}$ molar sodium bicarbonate should be given by mouth. If the patient is vomiting, it may be administered intravenously. A urinary output of 25 c.c. per hour should be maintained. An alternative formula is 1 c.c. of plasma per kilo for each per cent of body area burned. An equal amount of electrolyte solution is given plus 2,000 c.c. of 5 % glucose in water during the first 24 hours. On the second day plasma and electrolyte solution are reduced 50 %. On the third day no plasma is given and fluids are limited to normal amounts to avoid pulmonary edema. The severity of shock may be reduced by giving plasma as soon after the burn as possible. One-half the required amount should be given in six hours since extravasation of plasma about the burned area occurs early, and resorption of edema fluid usually begins in 36 to 48 hours. In the event of catastrophe, plasma substitutes or expanders such as

dextran or polyvinyl pyrrolidone may be used. Dextran is a polysaccharide carbohydrate hydrolyzed to smaller molecules. It is administered in 6 % solution and remains in the blood for about 36 hours. The clumping and rouleau formation of red cells caused by dextran administration may complicate blood grouping. As well as plasma it is advisable to give whole blood beginning on the third day. Evans has obtained excellent results in the recovery from shock, in speeding skin grafting, and in decreasing the period of hospitalization by the use of whole blood. This procedure is advantageous in combating the marked secondary anemia due to the destruction of red blood corpuscles by the burn.

3. Oxygen should be administered by nasal catheter, mask or tent. If there is evidence of burns of the upper respiratory tract with obstruction of the larynx, tracheotomy may be required. Pulmonary damage due to irritating gas or vapor may necessitate replacement of plasma and electrolyte therapy in varying degrees by whole blood. Reduction in the total amount of fluid administered may minimize the danger of pulmonary edema.

4. The use of adrenal cortical extract, testosterone and more recently, corticotropin and cortisone have been advocated in the treatment of severe thermal burns. Conflicting results have been reported in the use of all these hormones and further experimental and clinical studies are necessary before final evaluation may be determined.

5. *Antibiotics*—Penicillin which is most effective against gram-positive organisms has been used with excellent results. It should be given intramuscularly as soon as possible after injury to prevent invasion of the burned area by pathogenic organisms. A burn of 10 to 15 % may be treated by 100,000 units daily, while a burn of 30 % and up will require 500,000 to 1,000,000 units daily. Chloromycetin and terramycin, antibiotics with a broader spectrum of action than penicillin against both gram-positive and negative or-

ganisms, are advocated. Cultures should be taken at the first change of dressing, and the antibiotic therapy adjusted accordingly, depending upon the type and resistance of the organisms found.

Nutrition.—As a result of lowered food intake, deranged nitrogen metabolism, fever and infection, an individual suffering from extensive burns loses weight rapidly and there is marked wasting of muscles. To combat this, a diet high in protein, calories and vitamins should be given as soon as tolerated. A suitable diet consists of 150 to 200 grams of protein, 250 to 300 grams of carbohydrate, and 100 grams of fat. Sulfur-containing foods should be provided as well as large amounts of vitamin C and vitamin B complex. If vomiting is present in the early stages, intravenous therapy must be substituted about the third postburn day. Blocker has employed a small pump to force a semifluid diet continuously through a nasal catheter which is inserted into the stomach. Mildly burned patients can tolerate a higher food intake than those severely burned.

Local Therapy.—The total area of a burn and some estimate of depth of injury should be carefully recorded, since this determines to a large degree the amount of plasma and electrolyte therapy administered. The fact should never be forgotten that treatment of the general condition of the patient takes precedence over local care, unless two teams are immediately available. Failure to observe this rule has permitted many patients to slip into irremediable shock while a perfect dressing was being applied. If at all possible, the patient should be taken to an operating room where, under aseptic conditions, intravenous plasma is started, the burned areas are cleansed and application of a dressing is carried out. Adequate doses of morphine are given but general anesthesia is not employed. After the clothing has been cut away, the burned area should be gently washed with white soap and water.

Blisters are opened, but not cut away and loose shreds of epithelium are removed. No hard scrubbing or unnecessary exposure of dermis is desirable. The ideal treatment should convert the burned area into a clean wound. An attempt is made to (a) preserve any viable epithelium; (b) provide action against any remaining bacteria and permit free drainage of plasma into the gauze dressing; (c) maintain uniform pressure in order to reduce extravascular escape and surface oozing of plasma; (d) splint the burned area to accelerate healing.

Despite the imposing list of over 70 methods of treating burns, it should be recognized that variations in treatment must be made because of the age of the patient, the location and depth of the burn, and the agent causing the burn. The burned surface is dressed with fine mesh gauze impregnated with petrolatum, or other bland ointment. After applying a number of layers of flat gauze dressings the whole is covered with a bulky layer of cotton waste which is held under moderate pressure by an elastic bandage or adhesive tape. Plywood or plaster splints may be applied to prevent movement, if desired. The dressing is not changed for 7 to 10 days, depending on evidence of infection. This should be carried out in the operating room, with preparations having been made either to debride necrotic tissue or to apply skin grafts. Various acids and enzymes have been advocated to speed removal of necrotic tissue by frequent application in wet dressings. Pyruvic and phosphoric acid as well as enzymes such as streptokinase, streptodornase, trypsin and collagenase have been tried. However, use of these agents may expose the patient to secondary bacterial contamination or cause damage to viable islands of epithelium. Surgical removal of slough followed by early grafting has many advocates.

Special Areas.—On the face, genitalia and perineum, antibiotic ointments on fine mesh gauze, or frequently changed saline dressings may be used since pressure dressings are not

Fig. 17.—Marked keloid formation in Malayan youth following third degree burns of neck. These were excised and after inserting many dart incisions in the edges of the wound to break up direct scar pull, thick split skin grafts were applied with good result



A



B.

Fig. 18—A. Deep third degree burn caused by concentrated sulphuric acid.
B Free range of movement following split skin grafting.

practicable. Since Wallace has revived the *exposure method of treating burns*, many surgeons prefer to use this method for treating these regions and in fact other parts of the body, which may be kept free from pressure contact with the bed by elevation or position. The area is cleansed, exposed on clean sheets and permitted to form a dry brown crust which forms in 1 to 3 days. Partial thickness burns heal in about 2 weeks. In areas where full thickness loss of skin has occurred, the crust sinks below the surrounding level and exudate collects beneath. These crusts should be removed and skin grafts applied to the granulating area. When the burned area is relatively small and obviously third degree, it is sometimes advisable to excise the burned tissue completely and to skin graft immediately. In selected cases, the period of hospitalization is reduced to a minimum.

Treatment of Granulating Areas.—Prior to skin grafting, the hemoglobin should not be less than 90 %, and transfusions should be administered as required to achieve this level. The wound should be prepared by the application of a pressure dressing which creates a firm red bed of granulations. One of the many antibacterial ointments may be used, or, if desired, the dressing may be kept wet with saline running through tubes incorporated in the dressing. The dressing should be changed at frequent intervals depending on the bacterial flora, amount of exudate, and state of the granulations. Before skin grafts are applied it is important to culture the wound and obtain information as to the type of bacteria present. Certain organisms such as *B. pyocyaneus* and *proteus* may interfere with the take of the skin grafts. Neomycin ointment 5 mg. per gram or in wet dressings of the same strength is effective in overcoming these organisms. Any granulating area over two inches in diameter is worth skin grafting. Even smaller wounds should be resurfaced when they are situated near joints, in areas where contractures may form, or on cosmetically important regions.

CHEMICAL BURNS AND INJURY

Following injury with strong acids and alkalis, the affected areas should be washed with copious quantities of water as soon as possible to dilute and remove the chemical before reaction with the tissue and heat production can occur. The use of appropriate neutralizing agents such as diluted acetic acid or sodium bicarbonate may be considered subsequently but should not be of such concentration that secondary injury will be caused to the eye. Powdered lime should be wiped away carefully before water is used. Routine burn treatment is then given.

Phosphorus powders should be brushed off before applying water to the part. Burning phosphorus in contact with tissue should be treated by plunging the part in water immediately. The heat of burning phosphorus and not the acids formed during combustion causes the deep tissue coagulation and damage seen in these burns. The phosphorus is then scraped off carefully. The area should then be washed with a solution of sodium bicarbonate (2 tablespoons to a pint of water) which is followed by a 1 % solution of copper sulfate which will coat any remaining particles with a protective layer of copper phosphide. Regular burn dressings are then applied. Following severe white phosphorus burns common in wartime, systemic therapy should be started immediately. Methionine should be given in glucose saline (100 mg. per liter) to minimize liver damage. Isotonic solution made up of two-thirds saline and one-third sodium bicarbonate should be given by mouth. Adequate amounts of calcium and vitamin K should be supplied.

Magnesium.—The intense heat at which magnesium burns, approximately 3,500° C., causes deep destruction should a flare or incendiary missile explode accidentally. Following removal of any remaining metal and grossly charred tissue, a burn dressing

A.



B

C

Fig 19—A and B Explosion of magnesium incendiary bomb in hand of factory employec, with charring of hand, extensive third degree burn of arm, face and opposite hand.

C Result following thick, split skin grafting of arm and face with scalp graft to reconstruct eyebrow. The exposed bones of the forearm were wrapped in a pedicle flap to withstand the pressure of an artificial hand

should be applied until final demarcation of necrotic tissue occurs and reparative treatment may be started

Mustard gas causes blistering and deep destruction of skin. Contaminated clothing should be removed and liquid such as mois-

ture on the skin should be absorbed by dry gauze. Any remaining gas or liquid should be neutralized by Dakin's solution. Subsequently the area should be washed with soap and water, dried, and a burn dressing applied.



Fig 20—A. Area of scalp necrosis with exposed bone following electrical burn.
 B. Defect in skull after removal of necrotic bone. Dura was intact.
 C and D. Appearance after transfer of pedicle flap from abdomen to the defect in the scalp and calvarium using the forearm as a carrier

Lewisite which contains arsenic acts rapidly and causes intense irritation of the skin, so all contaminated clothing should be removed and treatment started as soon as possible. Lewisite may be neutralized with hydrogen peroxide followed by washing with soap and water. Skin blebs which contain arsenic should be drained and regular burn dressings applied.

ELECTRICAL BURNS

Following a severe electric shock most patients are unconscious, and about three-quarters require artificial respiration. This must often be continued for some time and the heart must be functioning. Fibrillation or arrest of the heart requires treatment by cardiac massage within 5 to 10 minutes and may be attempted if conditions are favorable. A fibrillating heart must be stopped by a shock from the defibrillator and then restarted. Once the patient has been resuscitated, shock should be treated in the usual way to bring the blood values to normal levels.

The frequency of these burns is decreasing because of the increased safety of construction of various electrical appliances. They differ considerably from thermal burns in that necrosis of tissue follows the path of the current deep into the body and quite frequently involves muscle and bone since the blood vessels provide an excellent path for conduction of electricity. In severe electrical burns when there is surface charring and probably deep damage, it is advisable to wait until slough or sequestra have separated to avoid loss of salvable tissue and risk of hemorrhage. Granulating areas may be resurfaced with skin grafts or pedicle flaps as indicated. Extremities which have been charred or show incipient gangrene should be amputated before infective or toxic symptoms appear.

RADIATION INJURY

Acute Total Body Radiation.—Explosion of an atomic bomb by military intention may

be expected to produce about 15 % of casualties suffering from radiation injury alone. The problems of triage are complicated by the fact that the sensitivity of man to massive total body radiation is not known. It is generally estimated that 400 r constitutes L.D.₅₀ in man but some will succumb to 200 r while others will survive high dosage. Estimation of the amount of radiation received by a dosimeter is not completely reliable, since it or part of the body of the individual may be shielded. In general those beneath the hypocenter of the bomb may be assumed to have received a heavier dose of radiation than others a thousand yards or more toward the periphery. Clinically patients suffering from acute radiation syndrome may be divided into three groups:

Group 1.—These have received supralethal dosage and recovery is improbable. Vomiting starts immediately or in a few hours followed by diarrhea, prostration, and death in a few days.

Group 2.—Survival is possible and moderate amount of exposure has been received. Vomiting occurs on the day of exposure and subsides in 24 hours. There is a reappearance of symptoms in one to three weeks.

Group 3.—Sublethal dosage has been received and survival is quite likely. There is no vomiting on the day of exposure and only mild symptoms subsequently.

In those who have a chance to survive it is important to direct therapy toward the prevention or treatment of:

- 1 Infection
- 2 Hemorrhage, mainly due to profound thrombocytopenia.
- 3 Anemia due to hemorrhage, increased rate of destruction of red blood cells and lack of production of red blood cells by the bone marrow.

4. Disturbance of fluids, electrolytes and acid-base balance by anorexia, vomiting, diarrhea and fever. Although pre-treatment by various measures will reduce the mortality in acute radiation injuries, no specific therapy

■ available as yet which is effective, when administered *following* exposure. The only methods available at present are antibiotics, blood transfusions and other supportive measures until regeneration of bone marrow occurs.

Acute Localized Radiation Injury.—Individuals employed in atomic energy plants or exposed to radioactive substances, x-ray

machines, or fluoroscopes in hospitals or industrial laboratories may receive large doses of ionizing radiations over a limited area of the body. The hands are particularly liable to suffer. Early symptoms are edema and erythema which become more intense in a few days. Vesicles form and may coalesce to form large blchs. Moderate or severe pain may be present. The injured area

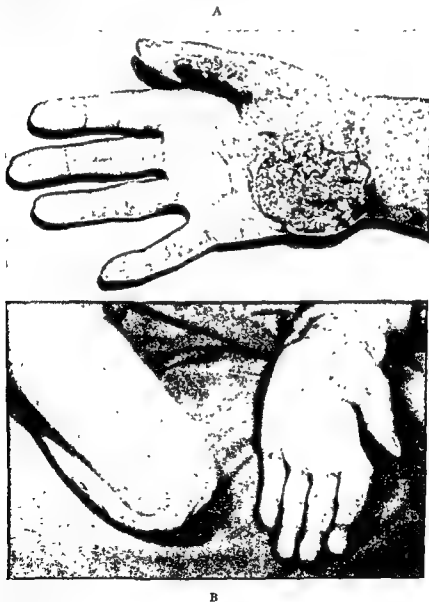


Fig. 21 —A X-ray therapy received 15 years previously for eczema of the hands followed by development of squamous cell carcinoma which necessitated amputation.

B Healed amputation stump, with the other hand showing ulceration which will probably progress to neoplastic change

should be covered with a bland ointment gauze and dressing which reduces pain. Infection may be prevented by adequate antibiotic therapy. Depending upon the dosage of radiation received and the resistance of the tissues, the skin and deeper tissues may heal or slough. Separation of slough should be awaited, when skin grafts may be applied. Thus an apparently doomed finger may be saved from amputation. However, because of the effect of radiation in causing sclerosis and obliteration of blood vessels, the injured skin will undergo more rapid atrophic changes than is the case with small dosage or chronic exposure to radiation. To avoid ulceration or neoplastic changes, it may become necessary to excise all degenerating areas and "recoat" them with a fresh healthy thick skin graft.

Chronic Radiodermatitis.—This may result as a sequel to acute injury or from the cumulative effect of repeated small doses of radiation. Telangiectasia, atrophy of skin, depigmentation, ulceration or keratoses may be present in any combination. Cancer may finally appear. It should be stressed that

such areas should be excised and skin grafted before neoplastic changes have developed.

Ultraviolet Radiation.—Exposure to ultraviolet rays may cause a first degree burn, and if very extensive will result in shock and in some instances death. Extensive sunburn may be treated by bland ointments. Constant exposure to sunlight over a period of years, especially in blondes, may give rise to sailors' skin, a condition which resembles chronic radiodermatitis, and which not infrequently results in neoplastic changes in the skin.

REFERENCES

- Berkow, S. G.: Value of Surface-area Proportions in Prognosis of Cutaneous Burns and Scalds, *Am. J. Surg.* 2: 315-317, 1931.
 Cope, O., Langohr, J. L., Moore, F. D., and Webster, R. C., Jr.: Expeditious Care of Full-thickness Burn Wounds by Surgical Excision, *Ann. Surg.* 121: 301-313, 1945.
 Evers, J. L.: *Textbook of Clinical Surgery*, 345.
 Harkins, H. N.: Treatment of Burns, Springfield, Ill., 1942, Charles C Thomas, p. 457.
 Walker, J., Jr., and Shenkin, H.: Studies on the Toxemia Syndrome After Burns, *Ann. Surg.* 121: 301-313, 1945.
 Wallace, A. B.: Treatment of Burns, *Ann. Roy. Coll. Surgeons England* 5: 283-300, 1949.

INJURIES DUE TO COLD

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FROSTBITE AND IMMERSION FOOT

While isolated cases of frostbite are relatively common in cold climates in civilian life, it is only in military campaigns that large numbers of cases are seen. This can be a matter of grave concern, and history records the bitter price paid by armies unprepared to meet the exigencies of winter warfare. The casualties from cold injuries may far outnumber those from enemy action. There is often difficulty in providing adequate clothing and shelter, and for evacuating the wounded. Many must remain immobile for long periods in cold and wet positions pinned down by enemy fire. At sea, survivors from sunken ships may sit for days or weeks huddled in lifeboats with their

limbs immersed in icy waters and their bodies exposed to winter weather. Besides the immediate loss of effective manpower, the victims of cold injuries may have sequelae that render them permanently unfit for service or incapacitate them for long periods.

Physiology and Pathology

The effect of cold on the tissues was described by Lake and most extensively studied by Lewis. Numerous workers since then have made substantial contributions. When an extremity is exposed to severe cold, vasoconstriction occurs evidenced by blanching of the skin. This may be followed by a bright pink color due to vasodilation and

lack of dissociation of oxyhemoglobin. Vasoconstriction then occurs and is permanent in the chilled state. All of the vascular tree is involved even to the major vessels. This appears to be a protective mechanism to conserve body heat, but it is at the expense of the exposed limb. The phenomenon of supercooling may prevent solidification until the temperature is considerably below that of the normal freezing point. As the temperature falls, the chilled or supercooled tissue becomes solidified although brittleness is not evident in the ordinary clinical cases.

The damage to the tissue depends on the severity of the cold and the duration of exposure. There may be injury from anoxia due to the vasoconstriction, and actual damage to the cells from the freezing process. The anoxic state is augmented by capillary stasis, especially as seen in chilled limbs without actual freezing where there is escape of the plasma leaving the red blood cells in a state of "sifting" or "sludging" as described by Kreyberg and Greene. In frozen tissue considerable damage may be caused by the formation of ice crystals that continue to grow disrupting the cells. This is not so evident in "quick freezing" as in a slower process. Experimentally, it has been shown that some tissues such as red blood cells may be frozen in glycerol and survive after thawing.

The thawing process is followed by a marked inflammatory reaction. Lewis suggested it resembled the "triple response" to histamine and thought the reaction might be due to the liberation of histamine-like products from the cellular injury. Thawing occurs either from without, in, or along the course of the vessels as circulation is reestablished. Edema soon forms with escape of plasma through the damaged capillary walls. This usually reaches its maximum in 48 hours. Stasis in the capillaries is apparently due to simple loss of plasma, possibly to the action of cold agglutinins or abnormal adhesiveness of the red blood cells. The sur-

face temperature of the part is raised considerably, probably due to the opening of arteriovenous channels. These channels may rob the injured part of adequate circulation as gangrene will sometimes develop in a part apparently well supplied with blood.

As the edema increases, areas of cyanosis may appear, and there is always danger of infection in the devitalized areas.

There are many factors that modify the actual injury such as the degree of cold, the duration of exposure, the surrounding medium, the resistance of the individual, and the effects of fatigue, emotion and malnutrition.

Many terms have been used to describe the syndrome produced by exposure to cold such as frostbite, immersion foot, trench foot, chilblains, etc.

Frostbite.—This term is used to describe the condition resulting from exposure to cold of sufficient severity and duration to produce ice in the affected parts. In frostbite there is vasoconstriction that can be relieved occasionally by vigorous rubbing as is often seen in the ears. This will frequently prevent further injury as the resultant hyperemia will enable the tissues to maintain an adequate temperature. When the defences are overcome, freezing occurs. Anesthesia is limited to the frozen area, thus differing from immersion foot, and edema does not occur until the part is thawed. The sequence of events is then similar to that of injury due to chilling or wet cold.

Immersion Foot (Trench Foot, Water Bite, Lifeboat Leg, Seaboot Leg).—This is the name given to a condition resulting from long exposure of the limbs in icy water or to a cold moist environment. The term is inadequate as the limb need not be immersed and the condition also occurs in other members.

The severity of the cold is not sufficient to cause freezing, but the conduction of heat from the tissues sets up a syndrome that has immediate and disabling sequelae. It resembles and is probably identical with the

trench foot of the first World War. It occurs chiefly in survivors from shipwrecks who are forced to sit in lifeboats, rafts or floats with their feet dangling in icy water. Cases have been reported occurring in warmer water

almost all the exposed part. They are first a livid color, which later changes to a pallid waxy appearance, and if the temperature rises slightly become mottled with blue and green areas. After rescue and exposure to



Fig. 22—Appearance of feet of patient 2 days after suffering immersion for 24 hours

but these were associated with poor nutrition and long exposure to brilliant sunlight in cramped positions. After some hours of exposure the feet, which are at first painful, become swollen and numb, the edema involving

room temperature the affected limbs pass through three stages: prehyperemia, hyperemia and posthyperemia.

Prehyperemic Stage.—At first the feet are numb, the patient feels as though he were

walking on blocks of wood, and he should always be assisted. The color begins to change from pallor to lividity and the legs show various color patterns. Vesicles and even large bullae appear. There is anesthesia of a stocking type with occasional hyperalgesic areas. The posterior tibial and dorsalis pedis arterial pulsations cannot be detected.

Hyperemic Stage.—At ordinary room temperature the feet quickly pass into the hyperemic stage. The limbs become hot to the touch and the color a livid red. The dorsalis pedis artery becomes palpable and bounding in character. The circulation is unstable. Congestion appears when the legs are in a dependent position and blanching when elevated. The pain at this period is intense and of a throbbing, burning character. This may last for several days and then change to a shooting, stabbing pain in the dorsum of the foot, radiating to the toes. This may last for several weeks. The edema increases, and large blisters are common, especially over the dorsum and malleoli. In severe cases, portions of the foot, usually the toes, remain discolored and cold, heralding the onset of gangrene.

Posthyperemic Stage.—The hyperemic stage soon merges in a few days into the posthyperemic stage. The burning shooting pains subside but the feet are subject to intermittent aching pain and edema readily appears when the feet are in a dependent position. The circulation is unstable as evidenced by color changes in different positions. Excess sweating is common and the patients complain of cold clammy feet. Walking is often difficult as proprioceptive sense appears deficient. The sensitivity to cold, hyperhidrosis and pain may persist for a long time and present a difficult problem in rehabilitation.

Treatment.—The aim of the treatment of frostbite or immersion foot is to prevent or minimize necrosis. This has been the subject of a large amount of experimental work and the interest of many military sur-

geons. It was thought for a long time that slow thawing or warming was essential to allow the vessels time to recover their tone and to limit the resulting hyperemia so that the increased metabolic demands of the injured tissues could be met by an adequate blood supply. To accomplish this, the limbs were rubbed with snow, packed in icebags and cooled with fans or special cabinets. Experimental work in the last few years has indicated that less tissue is lost by rapid thawing than by slow warming, and that stasis in the vessels is delayed. There is evidence that the larger vessels are in spasm during the chilling period and only relax when the tissue temperature is nearly that of body temperature. While the results of animal experiments cannot always be applied to man, it is probable that no harm is done by rapid rewarming of the frozen part to body temperature.

The blisters that have formed may be left intact, but if ruptured an occlusive dressing should be applied. Antibiotic drugs and antitetic serum should be given and sedation for pain when necessary. There is some evidence that anticoagulants may be useful in preventing thrombosis in the injured vessels, but should only be used when adequate control is available. Vasodilating drugs, lumbar blockage, or lumbar sympathectomy, have not been proved of value in the acute phase of frostbite nor in immersion foot where the sympathetic fibers seem to be inactive because of the cold injury. They may be tried, however, if properly supervised, as an occasional case is apparently benefited by the procedure. However, in the late sequelae of painful, sweating hands or feet, where the digits become tapering and shiny, and the movements restricted, sympathetic interruption is of considerable value. ACTH and cortisone have not been found useful.

Treatment must also be directed to keeping the injured parts scrupulously clean, to prevent any maceration between the toes

and to protect them from infection and injury. Necrotic tissue should be carefully débrided, but amputation in the noninfected cases should be delayed as long as possible. It is surprising how often one may be deceived in mistaking superficial necrosis for gangrene of a large part. Following definitive treatment, physiotherapy is a most valuable aid to rehabilitation.

REFERENCES

- Finneran, J. C., and Shumacker, H. B., Jr: Studies in Experimental Frostbite, Further Evaluation of Early Treatment, *Surg., Gynec. & Obst.* 90: 430-438, 1950.
- Greene, Raymond: Frostbite and Trench Foot, *Lancet* 1: 303-305, 1910.
- Kreyberg, Laev: Development of Acute Tissue Damage Due to Cold, *Physiol. Rev.* 29: 156-167, 1949.
- Lake, N. C.: Investigation Into the Effects of Cold Upon the Body, *Lancet* 2: 557-562, 1917.
- Lange, K., and Boyd, L. J.: The Functional Pathology of Experimental Frostbite and the Prevention of Subsequent Gangrene, *Surg., Gynec. & Obst.* 80: 346-350, 1945.
- Lewis, T., and Love, W. S.: Vascular Reactions of the Skin to Injury. Part III. Some Effects of Freezing, of Cooling and of Warming, *Heart* 13: 27-60, 1926.
- Ungley, C. C., Channell, G. D., and Richards, R. L.: Immersion Foot Syndrome, *Brit. J. Surg.* 33: 17, 1945.
- Webster, D. R., Woolhouse, F. M., and Johnston, J. L.: Immersion Foot, *J. Bone & Joint Surg.* 24: 785, 1942.
- White, J. C.: Vascular and Neurologic Lesions in Survivors of Shipwreck, *New Eng. J. Med.* 228: 211, 1943.

CHAPTER VII

ANESTHESIA

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INTRODUCTION

Attempts to relieve pain were made in very early times. Until the middle of the past century methods and drugs used were crude and results uncertain. Various agents were known to produce artificial sleep, and surgical operations were performed while patients were under the influence of such drugs. Those used most frequently were henbane, poppy (opium), mandragora, and hemp.

Many of the anesthetic agents now in use were known to chemists long before anyone appreciated their anesthetic properties. Ether, for example, was discovered early in the thirteenth century and was called "sweet vitriol"; but the first ether anesthesia of which there is authentic record was produced in 1842 by Crawford Long. Since that time, many other drugs have been found to produce safe anesthesia, so that now there are many agents and techniques available from which to choose the combination best suited to meet the needs of patient, surgeon, and anesthetist.

CHOICE OF ANESTHESIA

The anesthetic selected should provide the patient with a comfortable and not too unpleasant interlude, and the surgeon with as nearly ideal operating conditions as possible without increasing the risk to the patient.

The patient may have prejudices regarding anesthesia and his wishes must be given consideration, however, his physical status and the nature of the contemplated surgery are usually more important factors. The anesthetic drugs selected should be such that their action does not adversely affect other complicating diseases. For example,

patients with respiratory infections are usually safer if not given an inhalation agent, and patients in shock should not receive a spinal anesthetic.

The anesthetist should know the surgeon's preferences, how much relaxation he will require, and how long he is likely to take to perform the operation. For most intra-abdominal operations, it is necessary to provide a marked degree of relaxation, whereas for a mastectomy a less potent anesthetic agent or a lighter level of anesthesia suffices.

In a doubtful case, the anesthetist should select an agent and technique with which he is thoroughly familiar, rather than try one which theoretically might be better but with which he has had little or no experience.

GENERAL CONSIDERATIONS

During the administration of an anesthetic, powerful drugs are employed to an extent approaching the limits of toxicity. It is therefore important to recognize the signs of adequate dosage and of overdosage. One of the prime requirements of an anesthetic drug is that its effect shall be reversible and its toxic action minimal.

Analgesia means loss of sensation to pain without unconsciousness.

Anesthesia means loss of all sensation. Under general anesthesia there is also loss of consciousness. Anesthesia may be of the conduction or general type.

General anesthesia may be produced by a number of different drugs and administered to the patient by several different routes, respiratory, intravenous, or rectal. The route and technique of administration are dependent upon the physical characteristics of the drug used. Regardless of the route of administration, the drug must be carried

by the blood stream to the central nervous system before any signs of general anesthesia become evident. Since different parts of the central nervous system are affected by different concentrations of the anesthetic drug, it is possible to produce safe levels of anesthesia without too much depression of the vital centers. Generally speaking, the more vital the center, the greater the concentration of drug necessary to paralyze it.

First Stage: Stage of Induction and Analgesia. It is characterized by a gradual loss of pain sense and consciousness.

Second Stage: Stage of Delirium. During this stage the patient is unconscious but may still react reflexly to various stimuli. The reflexes become progressively diminished but muscle tone is still present. Control of the higher cerebral centers is lost but the lower centers are relatively unaffected. It

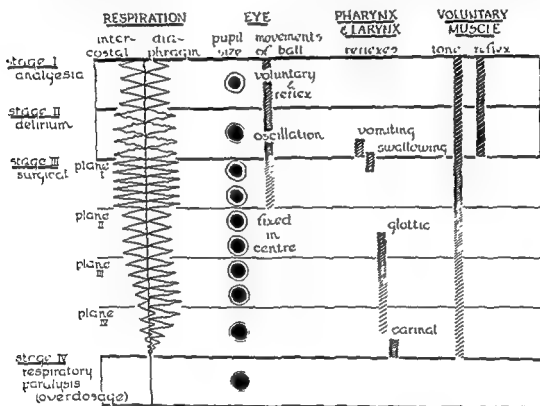


Fig 23.—Stages of anesthesia

The various stages of anesthesia represent a progressive diminution of irritability and conductivity culminating in complete inhibition or paralysis of the different parts of the central nervous system until respiratory and cardiac arrest occurs. All the stages and planes of anesthesia pass gradually from one into the next with no sharp line of demarcation. The most commonly used system for describing the level or degree of anesthesia is that of Guedel. In it there are four stages. The third stage, or stage of surgical anesthesia, is divided into four planes.

is during this stage that the patient may react violently to any real or imagined stimulation.

Third Stage: Stage of Surgical Anesthesia. During this stage sympathetic and voluntary muscle reflexes, including respiratory reflexes, disappear and muscle tone decreases. There is progressive paralysis of the muscles of respiration. Respirations are regular and their rhythm is automatic in type.

Plane 1: The eyelid and swallowing reflexes are absent. The eyeball usually oscil-

lates but may become fixed eccentrically; breathing is regular

Plane 2: Orbital movements have ceased and the pupils begin to dilate. Respiration is still regular but is little shallower than in Plane 1.

Plane 3: Activity of the intercostal muscles decreases. The depth of respiration is less and the inspiratory phase is shorter. Pupillary dilatation increases

Plane 4: The intercostal muscles are completely paralyzed, so that the movement of the chest and upper abdomen develops a seesaw characteristic because the diaphragm is the only respiratory muscle still functioning and the upper part of the chest is drawn in during inspiration. The respiratory exchange is greatly reduced

Fourth Stage: Stage of Overdosage. There is respiratory arrest which is soon followed by cardiac arrest unless artificial respiration is instituted and the drug eliminated. The pupils are widely dilated.

SIGNS OF ANESTHESIA

These are the physical signs which indicate the degree of activity, inhibition, or paralysis of the various parts of the nervous system. They will be considered under the following headings: sensory, motor or muscular, respiratory, ocular, and circulatory

Sensory signs disappear early and are manifest by breath-holding, vomiting, swallowing, and moving. The patient may voluntarily hold his breath during the first stage, or, because of reflex stimulation, during the second stage. Movements occur in a similar manner. Vomiting takes place just as the second stage is passing into the third stage, first plane, and swallowing in the upper first plane.

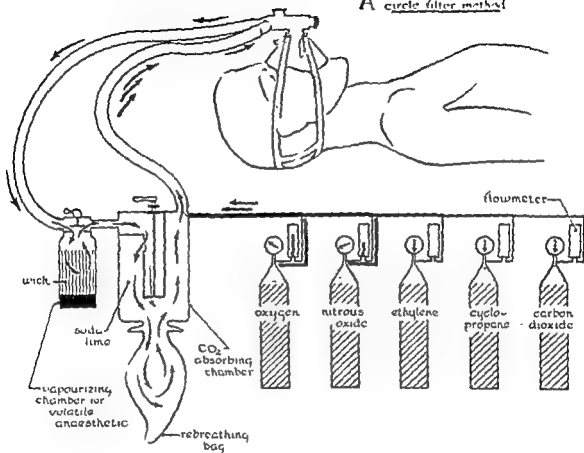
Muscular or motor signs are very important, especially to the surgeon. During the first stage, voluntary muscles are activated either by the will or by reflex stimulation. During the second stage, voluntary control

of movement is lost but reflex responses persist and movement may be violent. The eyelids and jaws resist opening. Skeletal muscles are rigid. During the third stage there is no response to sensory stimulation and muscle tone becomes progressively weaker. In the fourth stage there is complete muscular flaccidity.

Respiratory signs. The character of respiration undergoes several changes with different levels of anesthesia. During the first stage, breathing is under voluntary control and thus may be modified either by the will or reflexly. Breath-holding, rapid breathing, and coughing occur. During the second stage, breathing is affected by sensory stimuli and is quite irregular. The irregularity gradually diminishes until the third stage is entered, at which point respirations become regular and automatic. In this stage, inspiration and expiration are equal in time, amplitude, and force. There is no pause between them. As the intercostal muscles become paralyzed, chest movements diminish until expansion fails to occur during inspiration. Inspirations become shorter, quicker, and gasping, while the expiratory phase is lengthened, less forceful, and followed by a gradually lengthening pause until respiration ceases.

Eye signs are of considerable value in estimating the level of anesthesia. The pupils, however, are affected by preoperative medication; atropine dilates and morphine constricts them. During the first stage the eyelids and eyeballs move both voluntarily and reflexly. The pupils may be dilated if the patient is frightened. During the second stage the lids are rigid, the eyeballs moving or eccentric, and the light reflex very active. During the third stage there are no voluntary movements. In Plane 1 the lids are relaxed, the eyeballs moving or eccentric, and the pupils constricted. In Planes 2, 3, and 4 the eyeball is centrally fixed, and the pupils become dilated as the light reflex is gradually lost.

A circle filter method



B two filter method

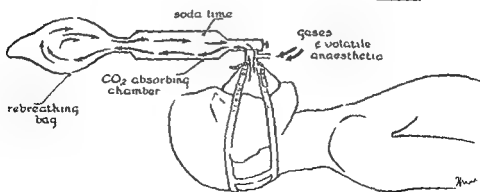


Fig 24 —Anesthesia Apparatus.

The circulation does not show changes which can be considered characteristic of the different levels of anesthesia, except for its profound depression which accompanies overdosage. Cyanosis appears only when the patient is receiving insufficient oxygen and is a condition which should never be allowed to continue. Pallor may be seen in the patient who is anemic or in shock in which case it should be treated by the administration of whole blood. The pulse of the patient under anesthesia varies greatly. It is frequently more rapid than normal before induction. This is due in part to excitement and in part to the use of atropine or scopolamine, which tend to paralyze the vagus and leave the cardiac accelerating nerves unopposed. As the level of anesthesia deepens into the third stage the effect of excitement is lost.

Since the various stages and planes of anesthesia pass into one another so gradually, it is necessary to consider together several of the above signs in order to determine accurately the level of narcosis.

GENERAL INHALATION ANESTHESIA

There are several available techniques for administering inhalation anesthesia. These depend upon the drug used, equipment available, experience of the anesthetist, surgical requirements of the case, and, to a certain extent, climatic conditions.

The simplest is that known as the *open drop method*, which requires very little equipment. The mask or cone consists of a framework of wire over which gauze or cotton is stretched. This is held over the mouth and nose of the patient while the anesthetic agent is poured upon it. The patient breathes through the mask inhaling some of the anesthetic agent. With this method a high percentage of the drug is blown away with each expiration and there is very little accumulation of carbon dioxide beneath the mask. When ether is used it is frequently

difficult to anesthetize a resistant patient without soaking the mask so thoroughly that the fluid will run onto the patient's face or eyes producing burns.

A modification of the open method is referred to as *semiclosed*, in which the mask is wrapped with towels in such a manner that some of the exhaled carbon dioxide and anesthetic agent are retained and rebreathed, permitting a greater concentration of the drug under the mask.

Other methods require more equipment and are more difficult to master but are used because they enable the anesthetist to provide more satisfactory operating conditions with less disturbance to the patient. Each employs a *closed system* comprising a *rebreathing bag* and an absorbing chamber for the elimination of excess carbon dioxide.

The simplest apparatus for *closed inhalation anesthesia* consists of a face mask, ether chamber, and rebreathing bag or chamber, which is filled with air or oxygen. This type of apparatus is not very satisfactory, since carbon dioxide is permitted to accumulate and if adequate oxygen or air is not added anoxia will result.

A modification of this system uses nitrous oxide and oxygen together with ether in a continuous flow at a rate which provides satisfactory levels of carbon dioxide and oxygen. Though reasonably efficient, this method is wasteful of gases and ether.

A further modification of the closed system is the *carbon dioxide absorption technique*. By this method oxygen requirements are maintained while carbon dioxide is removed by some chemical agent such as soda lime. Economy in the use of anesthetic agents is provided and loss of body heat and fluid through exhaled air is diminished. Control of the level of anesthesia is simplified and patients may be carried for longer periods without serious physicochemical upset.

The modern anesthesia *gas machine* consists of a number of parts in a mobile unit: (1) a framework on wheels to support cylinders of gas, flow meters, absorbers, etc.,

(2) yokes for attachment to cylinders of such gases as oxygen, nitrous oxide, cyclopropane, carbon dioxide, helium, and ethylene, (3) flow meters to control the administration of the various gases, (4) a bottle or other container with a wick to serve as a reservoir and vaporizing chamber for volatile liquids, (5) a bag or balloon for purposes of rebreathing and artificial respiration, (6) a carbon dioxide absorbing chamber which is filled with soda lime or Baralyme. This must not be used if trichlorethylene is the anesthetic agent.

There are two types of soda lime chambers in common usage. The *to-and-fro* type is placed near the face mask, between it and the rebreathing bag. There are no valves used with this type of absorber, and the anesthetic mixture passes through it twice with each respiratory cycle. The *circle filter* type of absorber is equipped with valves to keep the anesthetic mixture moving in only one direction.

(7) Flexible tubing to connect the patient to the machine, (8) a face mask with suitable connectors. The face mask has a rubber cushion, so that it fits tightly against the face without excessive pressure. The mask is held in place by suitable retaining harness. During endotracheal anesthesia the face mask is removed.

In the anesthetic gas machine, there is no motor or pump. The anesthetic mixture is propelled by the respiratory efforts of the patient, except in special circumstances when the anesthetist breathes for the patient by rhythmically compressing and relaxing the breathing bag. *Assisted or compensated respiration* is to be performed when natural respirations are unduly depressed, as in patients submitted to curare or deep anesthesia. Administration of any excessive anesthetic agent must be avoided in the latter instance. *Controlled respiration* is the term generally used to describe the technique when the anesthetist purposefully produces apnea in the patient and then controls the rate and depth of respiration. It may be employed in tho-

racic surgery and in abdominal operations, when a very quiet, relaxed field is desired for short periods of time.

The preceding are all described as inhalation techniques, since the anesthetic vapor reaches the patient because of his own inspiratory efforts. Another method is that of *insufflation*, in which the anesthetic vapor is blown into the patient's respiratory system. An air compressor forces air over the surface of, or bubbles it through, the anesthetic agent and thence to the pharynx and larynx of the patient. This technique is in less frequent use than that of the inhalation type. It may be used in nose and throat operations.

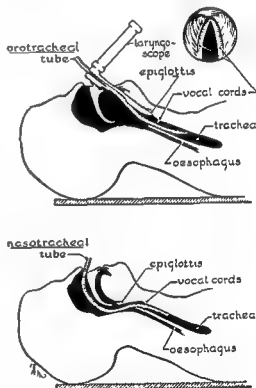


Fig 25—Endotracheal anesthesia

In *intratracheal* or *endotracheal* anesthesia an artificial airway is inserted directly into the trachea. This maneuver requires experience. It may be performed blindly or under direct vision. The ability to intubate a patient easily may be a factor of lifesaving importance.

Some endotracheal tubes are stiff. Others are soft and flexible. It is most important that the lumen of the tube be of adequate size and its walls sufficiently rigid to prevent collapse and respiratory obstruction.

Usually, the anesthetist uses a laryngoscope to expose the glottis before passing the tube between the vocal cords. The laryngoscope must be held firmly but handled gently to prevent injury to the soft tissues of the patient's throat and to his teeth. Before tracheal intubation is attempted, the subject must be well relaxed.

Intratracheal anesthesia is maintained by either inhalation or insufflation methods. For inhalation the tube must be large enough to permit unobstructed breathing. For insufflation, since the patient must exhale around the tube, its cross-sectional area should be less than one-half that of the glottis.

VOLATILE ANESTHETIC AGENTS

Chloroform. This is a clear, colorless liquid at ordinary room temperatures and pressures and has a characteristic sweetish odor. The vapor is considerably heavier than air and is noninflammable. Chloroform boils at 140.2° to 141.1° F. The U.S.P. preparation contains a trace of alcohol to convert any phosgene which may be present into harmless diethyl carbonate. Chloroform, in the presence of an open flame, is converted into phosgene.

Chloroform may be administered with very little equipment, as in the open drop technique, or it may be given by anesthesia gas machine, which provides a greater quantity of oxygen for the patient.

With chloroform it is possible to produce excellent anesthesia with quiet respirations. Induction is pleasant and rapid, and any desired depth of anesthesia can be produced quite quickly. However, the margin of safety between third-stage anesthesia and respiratory and cardiac arrest is not great. Because of its direct toxic action, augmented by anoxemia, chloroform may cause serious

heart and liver damage. Death may result from ventricular fibrillation, since chloroform sensitizes the heart to the action of epinephrine. It is important therefore not to administer Adrenalin or similar compounds to patients before or during chloroform anesthesia. Some measure of liver protection is achieved by a preoperative diet rich in carbohydrates and amino acids and by the use of high concentrations of oxygen during the operation.

In tropical zones, chloroform is used frequently because it is less volatile than ether. It is still used in obstetrical anesthesia, and is especially effective in this field because it requires very simple equipment and a few drops will quickly bring relief during labor pains. Even in obstetrics, one must guard against liver damage by providing an adequate airway and avoiding deep levels of anesthesia.

Ether. Diethyl ether is the most frequently used of all anesthetic agents. It is a transparent, colorless, volatile liquid which unfortunately is explosive and inflammable. With ether it is possible to produce adequate muscular relaxation for any surgical procedure. Ether may be administered by the open drop, semiclosed, inhalation, or insufflation techniques, or by rectum.

Induction of anesthesia by means of ether is unpleasant, so that some other agent is usually used for this purpose; e.g., vinylene, ethyl chloride, chloroform, cyclopropane, or nitrous oxide, an intravenous barbiturate, or a rectal agent such as paraldehyde or Avertin.

Ether causes certain metabolic changes. Acetonuria, hyperglycemia, albuminuria, and a raised blood urea concentration are frequently noted after its prolonged administration. Liver damage, however, is usually slight and transient.

Trichlorethylene or Trifene is a volatile liquid with an odor somewhat like that of chloroform. It does not vaporize readily enough to be given by the open-mask method and must be administered from some form

of vaporizer bottle. The sodium hydroxide component of soda lime will react with it to form toxic products so that Trilene may not be used in a closed circuit. The greatest advantage of Trilene is that it produces marked analgesia in light anesthesia. It is employed as a supplement to nitrous oxide and oxygen. It is useful in obstetrics, dentistry, and in many minor operations such as the changing of painful dressings where analgesia rather than relaxation is required. It is not satisfactory for producing relaxation because in higher concentrations it usually causes marked acceleration of respiration. When Trilene, with or without nitrous oxide and oxygen, is used for the purpose of analgesia, it does not seem to have any toxic effects and the incidence of nausea and vomiting is very low. Induction and recovery are rapid and analgesia often persists following return of consciousness because Trilene is slowly liberated from the body fat.

Ethyl chloride is a colorless liquid with an odor somewhat like that of chloroform but more disagreeable. Because of its low boiling point (53.6° to 55.4° F), it must be kept in sealed glass or metal tubes.

Ethyl chloride may be used for the induction of anesthesia or as the sole agent for very short procedures. Induction with ethyl chloride is very rapid and may be complicated by laryngospasm. If its administration is improperly continued, respiratory arrest and death may occur with little warning. An abundance of air or oxygen should be given together with ethyl chloride and deep levels of anesthesia must not be attempted. If the period of anesthesia is short, recovery is rapid and untoward effects are not seen.

Nitrous Oxide. This was one of the first agents to be used for general anesthesia and is still popular. At ordinary temperatures and pressures it is a gas and must be dispensed in steel cylinders and administered from a gas machine. It provides speedy induction but the level of anesthesia produced is limited to Plane 1 of Stage 3. It does not

therefore produce satisfactory muscular relaxation and is seldom used for operations involving the peritoneal cavity unless some stronger agent such as ether or cyclopropane is added. When accompanied by an adequate supply of oxygen, nitrous oxide has very little effect on metabolism and recovery from anesthesia is rapid. It is frequently used in obstetrics since it gives relief from pain during contractions and quick recovery thereafter.

Ethylene. This is a hydrocarbon constituent of illuminating gas. It has a pungent, sweetish odor and is inflammable. Compressed in steel cylinders, it is administered by means of a gas machine and mask. Ethylene is a more potent agent than nitrous oxide but is still not strong enough to produce suitable relaxation in all cases unless ether is added to the mixture. Nausea and vomiting are more common following ethylene than nitrous oxide anesthesia.

Cyclopropane. This is a cyclic hydrocarbon which is nonirritating to the tissues. It has a peculiar odor somewhat like garlic. Induction with cyclopropane is rapid and not unpleasant. Several methods of administration are employed. At the outset, the patient may be given pure oxygen to which cyclopropane is gradually added until the desired level of anesthesia is reached. A mixture of cyclopropane and oxygen may be turned on before the mask is applied to the patient's face. To an initial mixture of nitrous oxide and oxygen, cyclopropane may be added.

Irrespective of the method used, it is essential to watch the patient with great care to avoid respiratory arrest. Cyclopropane is usually administered by the closed carbon dioxide absorption technique. The gas is expensive and inflammable and should not be allowed to escape into the atmosphere of the operating theater. By experienced anesthetists cyclopropane may be used for the controlled respiration technique of Guedel. The patient is rapidly brought to a state of respiratory arrest by high con-

centrations of cyclopropane associated with carbon dioxide absorption at a level below that necessary to stimulate respiration. By this means it is possible to provide quieter operating conditions and better oxygenation of the patient than when active breathing is taking place.

Cyclopropane is a much more powerful agent than the other gas anesthetics. It is not as potent as ether or chloroform but in most cases produces adequate relaxation. When necessary, ether may be added to the mixture or additional relaxation may be provided by curare.

Though relatively nontoxic, cyclopropane sensitizes the heart muscle to Adrenalin and similar compounds, so that ventricular fibrillation may result. For this reason, sympathomimetic drugs, with the exception of Neo-Synephrine, should not be given with cyclopropane.

RECTAL ANESTHESIA

At present, rectal anesthesia is seldom used to produce more than basal narcosis. Previously it was occasionally relied upon for total anesthesia.

The following drugs may be used for rectal anesthesia: ether dissolved in oil, tribromethanol dissolved in amylene hydrate (Avertin), trichlorethanol, paraldehyde in oil, and certain barbiturates (Evipal or Pentothal Sodium).

Ether has been administered rectally for almost as long as it has been used by inhalation. Early attempts were largely unsatisfactory because insufficient dilution of the ether led to mucosal irritation. The dosage of rectal ether is 0.75 to 1.0 c.c. per pound of body weight, the total not to exceed 160 c.c. The solvent may be olive oil or some other bland oil in a mixture containing 50 to 65% of ether.

Avertin is frequently used as a basal anesthetic, especially for nervous subjects such as toxic thyroid cases or children. A 25 to 35% solution of Avertin with amylene hy-

drate in distilled water warmed to 40° C. is administered after first testing it with a drop of Congo red. If a blue color develops the solution should be discarded. The dose varies from 50 to 100 mg. of Avertin per kilogram of body weight. The effect is usually noted within 10 minutes and reaches a maximum in 25 to 30 minutes. Though Avertin may be employed as a total anesthetic, the margin of safety is thereby greatly reduced, so that it is preferable to supplement basal Avertin anesthesia by means of nitrous oxide, cyclopropane, or ether.

Chloral hydrate and paraldehyde are seldom used as rectal anesthetics. Evipal or Pentothal, dissolved in distilled water, is frequently used rectally in children. The dose is 0.1 Gm. per year of age up to a maximum of 2.0 Gm.

INTRAVENOUS ANESTHESIA

The popularity of intravenous anesthesia was established during World War II and has since continued. The drugs most commonly used are the ultra-short-acting barbiturates, Pentothal Sodium and Evipal. Other agents, such as ether and paraldehyde, have not proved satisfactory. The advantages of intravenous anesthesia by the short-acting barbiturates are the ease of its administration, simplicity of required equipment, rapidity of induction and recovery, and absence of postoperative nausea and vomiting or other untoward sequelae.

In excessive amounts, intravenous barbiturates cause severe *respiratory depression*. *Laryngospasm* is a serious complication which occurs because the laryngeal reflexes are not depressed until the patient is quite deeply anesthetized. If the anesthetic is to last more than a few minutes, it is advisable to administer oxygen and to supplement basal intravenous anesthesia by nitrous oxide, cyclopropane, or ether.

Pentothal Sodium is administered as a 25% solution in distilled water. For adults, the total dose is 0.5 to 1.0 Gm. Unless

the trachea is intubated immediately, Pentothal should not be used for operations on the face, mouth, or throat, because of the frequent occurrence of laryngospasm in such cases

RELAXING AGENTS

Though not an anesthetic drug, curare has become an important addition to the anesthesiologist's armamentarium. It was introduced to clinical anesthesia by Griffith in 1942. Several preparations are now available that may be administered repeatedly without appreciable toxic effect. If curare is used, however, the anesthesiologist must be prepared to carry on artificial respiration. Fortunately, the diaphragm is less sensitive than other muscles to the action of curare.

The function of curare is to produce adequate muscular relaxation in the presence of relatively light inhalation or intravenous anesthesia. The dosage varies with the type of preparation. Maximum relaxation is attained within 5 minutes and lasts about half an hour. A patient receiving ether requires a much smaller dose of curare than when some other form of anesthesia is being administered. When a spinal anesthetic is wearing off, curare will provide satisfactory relaxation for closure of an abdominal wound. In the conscious patient, however, a light general anesthetic should precede the administration of curare since subjectively the effects of this drug may be terrifying.

COMBINED ANESTHESIA

The modern anesthesiologist frequently combines 2 or 3 types of methods of anesthesia in a single case in order to take advantage of the special benefits of each, as well as to reduce the ill effects of overdosage by one or other. Many surgeons do an abdominal field block even though the patient is to receive a general anesthetic. Such interruptions of nerve conduction in the field of operation greatly reduce the amount of general anesthetic required.

Spinal anesthesia may be supplemented by nitrous oxide, cyclopropane, or Pentothal. In this manner the completely relaxed but nervous patient may be rendered unconscious.

The combination of nitrous oxide and curare with Pentothal is suitable for almost any operation. Nitrous oxide provides analgesia, Pentothal loss of consciousness, and curare muscular relaxation.

RESUSCITATION

Before or during the course of an anesthetic it is frequently necessary to administer drugs or fluids to maintain circulatory tone. Most anesthesiologists believe that spinal anesthesia should be preceded by the administration of some analeptic drug in order to prevent a fall in blood pressure. Ephedrine, gr 1 to 1½, is given by hypodermic or intramuscular injection a few minutes before the anesthetic is injected into the spinal canal. Other drugs, such as Methedrine or Neo-Synephrine, may be used in place of ephedrine.

Glucose in water or normal saline, plasma, or citrated blood may be required to maintain the patient's fluid balance and to counteract the effects of blood loss during the operation. In all major cases an intravenous infusion should be started before or immediately after the administration of the anesthetic. At this time venipuncture is more easily performed and a saline infusion provides a route for the emergency administration of analeptic drugs or blood transfusion.

Respiratory arrest may be due to an overdose of anesthesia, muscle relaxants, sedative drugs, or to an excessively high spinal anesthetic. Before starting any anesthetic, the anesthesiologist should be prepared to administer oxygen under positive pressure whenever the need arises, and to continue artificial respiration for as long as may be necessary. The ordinary gas anesthesia machine is suitable for this purpose. By rhyth-

mical compression of the breathing bag artificial respiration can be maintained until respiratory function has returned. If respiratory depression is profound and likely to remain so for some time, it is advisable to pass an endotracheal tube to ensure a non-obstructed airway and to avoid inflating the stomach.

PREPARATION OF THE PATIENT FOR ANESTHESIA

The administration of an anesthetic must not be lightly undertaken. Induction of anesthesia by any general or spinal anesthetic is a major procedure.

It is generally advisable to prescribe a barbiturate to ensure sleep during the night before operation. The combination of a quickly acting barbiturate with one of delayed action is preferable.

At the time he examines the patient, the anesthetist may also prescribe certain drugs to be administered before the patient goes to the operating theater. The purpose of the preoperative medication is to calm the patient and to make easier the administration of the anesthetic by decreasing the amount of secretions in the respiratory tract. Morphine together with atropine or scopolamine (hyoscine) is administered by hypo-

TABLE V
PREMEDICATION FOR GENERAL ANESTHESIA (AFTER LEIGH)

| AGE | MORPHINE (GR) | WITH SCOPOLAMINE (HYOSCINE) (GR) | OR ATROPINE (GR) |
|-----------------|------------------|--|---------------------|
| 1 month or less | nil | 1/600 | 1/400 |
| 1-2 months | | | |
| 2-3 months | | | |
| 3-4 months | nil | 1/600 | 1/400 |
| 4-5 months | | | |
| 5-6 months | | | |
| 6-7 months | nil | 1/600 | 1/400 |
| 7-8 months | | | |
| 8-9 months | | | |
| 9-10 months | 1/108 | with 1/600 | 1/400 |
| 10-11 months | | | |
| 11-18 months | | | |
| 18-mo-2 yr. | 1/72 | with 1/450 | 1/300 |
| 2-3 years | 1/60 | with 1/450 | 1/300 |
| 3-5 years | 1/48 | with 1/450 | 1/300 |
| 5-8 years | 1/36 | with 1/300 | 1/200 |
| 8-10 years | 1/24 | with 1/300 | 1/200 |
| 10-12 years | 1/18 | with 1/200 | 1/150 |
| 12-14 years | 1/12 | with 1/150 | 1/150 |
| 14-18 years | 1/8-1/6 | with 1/100 | 1/100 |
| Adult | 1/4 | with 1/100 | 1/100 |

The above dosage should be administered by hypodermic injection 30 to 60 minutes before the posted hour of the anesthetic.

NEMBUTAL DOSAGE (AT BEDTIME, THE PREVIOUS NIGHT)

| | | |
|------------|-------|-------------------------|
| 1-3 years | gr ¼ | dissolved in corn syrup |
| 3-7 years | gr ½ | dissolved in corn syrup |
| 7-12 years | gr 1 | dissolved in corn syrup |
| 12 years | gr 1½ | dissolved in corn syrup |

The anesthetist should visit the patient prior to operation, elicit a history of previous illness and anesthetics, and decide what pre-operative medication and anesthetic technique are most suitable in the particular case.

dermic injection one-half to one hour before the anesthetic is scheduled to begin. The adult dosage is morphine, gr. ½ to ¼, with atropine or scopolamine, gr. 1/200 to 1/100. Such sedation may be supplemented by one or other of the barbiturates. The larger

doses recommended are given to healthy, robust, young males, and the smaller doses to fragile and older people. In patients over 60, it is advisable to use atropine rather than scopolamine.

The amount of preoperative medication depends to some extent on the anesthetic agent or method to be used. With nitrous oxide and ethylene larger doses of premedication are required. Prior to chloroform, ether, or cyclopropane anesthesia, the dose must be considerably reduced to avoid respiratory depression which prevents a concentration of anesthetic sufficient to produce adequate relaxation. Pulmonary complications are prone to follow respiratory depression.

If barbiturates are used preoperatively, a subsequent dose of intravenous Pentothal must be proportionally diminished. Adequate preoperative sedation should precede spinal anesthesia. By this means the patient is rendered less apprehensive and more cooperative. In addition the barbiturate factor helps to prevent toxic reactions which may be caused by the local anesthetic drug.

Some patients are sensitive to morphine. This fact should be ascertained before operation and morphine replaced by such drugs as Pantopon, Dilaudid, or Demerol.

Avertin or a solution of a short-acting barbiturate may be given rectally together with atropine or scopolamine, but without morphine, to produce basal anesthesia in very nervous patients or children. It should be administered about half an hour before the time scheduled for operation.

EXPLOSION HAZARDS

Many anesthetic gases and vapors are explosive and inflammable. This is potentially dangerous to the patient and the personnel of the operating theater.

Ether, Vinethene, ethyl chloride, cyclopropane, and ethylene are all inflammable. When mixed with oxygen, they will explode with considerable force. Explosions may be caused by static electrical charges, by a

cautery, by a spark or short circuit in any electrical appliance commonly used in operating rooms.

The risk of static sparks is minimized by maintaining a relative humidity in excess of 55% which serves to ground all people and equipment in the operating room. When this degree of humidity cannot be attained, an intercoupler system may be devised to eliminate differences in electrical potential.

The electric cautery or other electrical equipment should not be used within the open chest or within two feet of the anesthetic mask when an explosive mixture is being administered. The closed circuit rebreathing technique minimizes the chance of explosion since the humidity within the machine is high and there is little or no escape of anesthetic gases into the operating room. If an electric cautery must be used near the face the anesthetic mixture must be nonexplosive.

INHALATION THERAPY

It is frequently necessary to use gases in pre- or postoperative treatment. Oxygen together with carbon dioxide is most commonly used. Cyanosis which may be controlled by oxygen therapy should never be permitted to persist untreated.

The earliest signs of oxygen lack are an increased volume of respiration, tachycardia, and elevated blood pressure, associated with headache, nausea, and lassitude. Among the many factors which may cause oxygen lack are pulmonary complications, heart disease, obstruction of the respiratory passages by foreign bodies, hemorrhage, and excessive anesthesia or sedation.

Oxygen may be administered in a tent, by a face mask, or by nasal catheter. The advantage of an oxygen tent is that its atmosphere may be cooled and humidified. The concentration of oxygen within a tent is not so great as that which may be obtained by the use of a mask. In the treatment of pulmonary edema, oxygen under positive pressure may be given by means of a mask.

A catheter in the nasopharynx provides the simplest and least expensive method of increasing oxygen concentration in the inspired air. By this means concentrations of 35 to 65% may be obtained.

In the presence of severe abdominal distention high concentrations of oxygen (95 to 100%) should be administered through a mask. Oxygen will frequently relieve the distention by replacing inert nitrogen in the distended bowel.

THERAPEUTIC USE OF CARBON DIOXIDE

Carbon dioxide stimulates the respiratory center. A small increase in the percentage of carbon dioxide causes marked increase in the volume of respiration. Therapeutically, carbon dioxide is never administered alone, but as a 5 to 10% mixture with oxygen. Postoperatively a mixture of carbon dioxide and oxygen assures adequate ventilation of the lungs and helps to prevent atelectasis. This mixture is also of considerable value in the treatment of hiccough.

THERAPEUTIC USE OF HELIUM

Helium is an inert gas, found in minute quantities in the atmosphere, and is the lightest of all the elements except hydrogen. A mixture of 20% oxygen and 80% helium has a specific gravity approximately one-third that of air or oxygen. Since the movement of a gas past a constrictive orifice is inversely proportional to the square root of its molecular weight, inhalation of a mixture of helium and oxygen decreases the physical effort involved and therefore the dyspnea of patients with severe asthma or of those with obstructing lesions of the larynx or trachea. Helium-oxygen therapy is usually given by means of a mask or hood.

INTRAVENOUS PROCAINE

For many years it was taught that one of the greatest dangers of local or regional

anesthesia was the accidental injection of the drug into a blood vessel; that such injection would be followed by profound cardiovascular collapse and irritation of the central nervous system leading to excitement, convulsions, and possibly death. When such reactions occurred, they were controlled by the intravenous injection of a barbiturate and by analeptic drugs. During recent years, however, procaine solution has been used intravenously for a variety of conditions. It has been found to reduce reflex cardiac disturbances, sometimes seen under general anesthesia, especially in intrathoracic operations. It may be used as a diagnostic or therapeutic measure in certain traumatic, inflammatory, and spastic conditions, and also for the relief of pain in burns, postoperative discomfort, and the pruritus which accompanies jaundice.

In selected cases procaine is generally given as a 0.1% solution, in an intravenous infusion. The rate of infusion should not exceed 1 liter per hour, and the patient must be watched constantly for signs of overdosage, such as excitement, apprehension, nausea or vomiting, twitchings, or variations in pulse, blood pressure, or respiration. If such symptoms occur, the infusion should be slowed or stopped, and a barbiturate given intravenously if required.

For emergency use in the operating room, with the patient under general anesthesia, the recommended dose of procaine is 100 mg. in a 1% solution.

Though the mode of action of procaine administered intravenously is obscure, its usefulness has been amply demonstrated. Its primary effect is to produce analgesia together with sympathetic paralysis and vasodilatation; secondarily, its action is parasympathomimetic and antispasmodic.

CONDUCTION ANESTHESIA

Regional anesthesia is produced by applying a drug along the course of a nerve to abolish conduction distal to the site of appli-

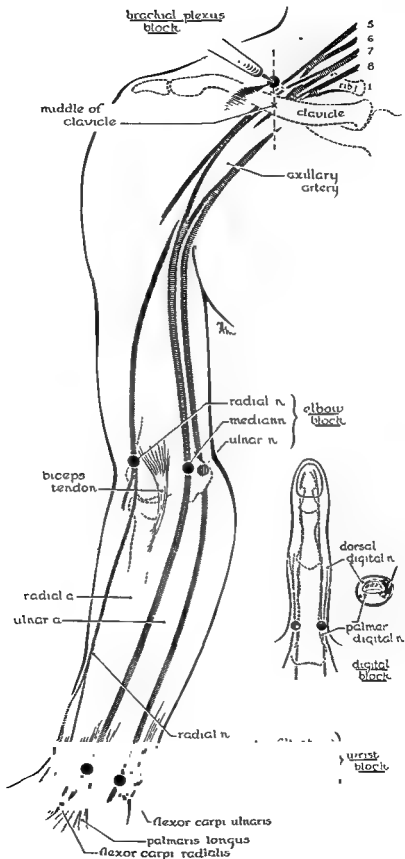


Fig 26—Regional nerve block anesthesia.

cation. It is classified according to the site of application.

1. **Topical.** The drug is applied to a surface where it anesthetizes nerve endings. This method is effective only on mucous membranes.

2. **Local Infiltration.** The drug is injected and affects the nerves with which it comes in contact. This method is applicable to minor procedures, such as the excision or biopsy of small tumors, or for suturing lacerations. A small amount of a vasoconstrictor such as epinephrine is usually added to prevent rapid absorption of the anesthetic drug and to diminish hemorrhage. Vasoconstrictors must be used cautiously or omitted in patients with heart disease or hyperthyroidism or in infiltrations of the fingers or toes.

4. **Nerve Block.** The nerves are blocked at any convenient point along their course to the periphery of the body before they have divided into their terminal branches. Efficient nerve blocking requires accurate anatomical knowledge. Any sensory nerve may be blocked but for many the landmarks are variable and difficult to define accurately.

5. **Paravertebral Block.** The spinal nerves are blocked as they emerge from the intervertebral foramina or in the vicinity of the vertebrae. A paravertebral block may be performed at any level of the spine and used for diagnostic or anesthetic purposes.

6. **Epidural Block.** The spinal nerves are blocked in the epidural space. This procedure may be carried out in the thoracic, lumbar, or sacral areas. It provides anal-

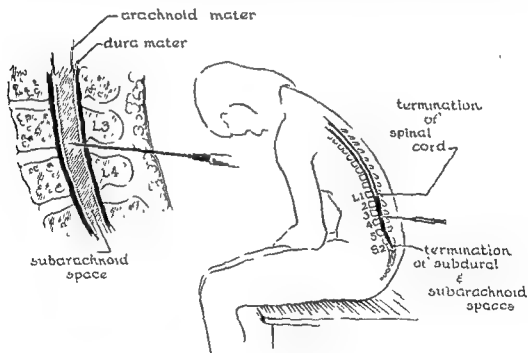


Fig. 27—Spinal anesthesia

3. **Field Block.** The terminal branches of the nerves are blocked by injecting a wall of local anesthetic along the borders of the area they supply. A continuous intradermal wheal is followed by gradual infiltration of deeper tissue planes to any required extent. Field block is used frequently in abdominal operations,

gesia similar to that produced by a spinal anesthetic except that motor and sympathetic depression is less profound. Sensory anesthesia, however, is complete and may be arranged to extend as high as the clavicles. Anesthesia resulting from epidural injection develops much more slowly than that produced by intradural administration,

7. Spinal Anesthesia. The spinal nerves are blocked within the dural space. Both anterior and posterior roots are affected. The extent of the anesthesia produced from a relatively small dose of anesthetic drug is much greater than can be obtained by a similar dose using any other regional technique. Spinal anesthesia can be used for operations anywhere below the clavicles, but is commonly reserved for operations below the diaphragm. It provides ideal conditions for abdominal operations or operations on the lower extremities, but should not be administered by an unskilled person without adequate supervision.

There is a great variety of drugs available for spinal anesthesia. Those most frequently used are procaine (Novocain), Pontocaine, Nupercaine, Metycaine, and Intracaine. The drug selected may be used either as a hypobaric, hyperbaric, or isobaric solution. The type of solution depends on the technique which is to be employed since most techniques of administration rely to a certain extent on gravity control of the range of spread of the anesthetic effects and to a lesser degree on the volume injected and rate of injection. The duration of their effects varies with different drugs, but with the same drug the duration may be affected by the concentration and total dose of the drug injected. The anesthetic effect following the administration of procaine is approximately one hour, of Pontocaine two hours, and of Nupercaine three hours.

In order to be able to provide spinal anesthesia of longer duration the *continuous* spinal technique was introduced by Lemmon. By this means a malleable needle or small catheter is left in the dural space and injections of the anesthetic drug are made at intervals during the course of the operation. Besides the obvious advantage of prolonging the anesthetic effect, this technique has other advantages over the single-injection technique.

A smaller initial dose may be used and, if signs of toxicity develop, it is pos-

sible to withdraw a moderate amount of the drug that has been injected. Any of the local anesthetic drugs may be used in the continuous spinal technique but procaine, Pontocaine, and Metycaine are those which are used most frequently.

The continuous technique may also be employed in a somewhat similar manner in epidural anesthesia, the most popular form being the continuous caudal technique which is used in obstetrics.

Advantages of Spinal Anesthesia.

1. Excellent muscular relaxation is provided.
2. There is little disturbance of metabolism.
3. The inhalation of irritating drugs is unnecessary so that the patient does not suffer from postanesthetic nausea and prolonged sleep.
4. The use of the electrocautery and other electrical appliances is permissible.

Disadvantages of Spinal Anesthesia.

1. It is difficult to control.
2. Its duration is variable and uncertain.
3. Technical difficulties may result in absence of anesthesia.
4. Motor anesthesia may ascend to high levels. If this occurs, respiration can and should be aided and adequate oxygen provided.
5. Severe circulatory collapse may follow the administration of a spinal anesthetic.
6. Consciousness during operation is not desirable in some cases.
7. Retching may follow traction on the viscera since the vagal pathways are not blocked.

II Postoperative : neurological complications may follow spinal anesthesia. The most common of these is headache.

Regional anesthesia is becoming more popular as techniques improve. This is true not only in providing anesthesia for surgery but, perhaps of more importance, in the various diagnostic and therapeutic procedures related to vasospasm, causalgia, etc.

REFRIGERATION ANESTHESIA

The numbing and pain-relieving effects of cold have been recognized for many centuries and amputations have been performed on limbs in a frozen or semifrozen state. Recently there has been increased interest shown in the use of controlled cold applied to the lower limbs to delay the progress of gangrene caused by obliterative vascular disease or trauma.

When cold is used prior to surgery the leg should be elevated slightly and ice bags placed in close contact all around the thigh in the region where the tourniquet will be applied. After 15 minutes, the thigh will be sufficiently numb to permit the application of the tourniquet tightly enough to occlude arterial circulation without causing discomfort. A thick layer of cracked ice is applied around the whole limb including the area of the tourniquet. After 1½ to 3 hours of cooling, the leg is insensitive and the sciatic nerve may be cut without producing pain or shock. Once the leg has been cooled sufficiently there should be no delay before commencing the operation since anesthesia will last only 20 to 30 minutes. The usual preoperative skin preparation is used. Following operation and removal of the tourniquet, the dressing is applied and the stump covered with ice bags to prevent a too rapid return to normal temperature. After cold treatment, healing may be delayed slightly but signs of shock and toxicity are greatly reduced and many patients in extremely poor condition have had amputations of gangrenous limbs performed with no anesthesia except that resulting from cold.

Another advantage of this method is that, when a patient is admitted in a very toxic state with a gangrenous limb, ice and a tour-

niquet may be applied and kept in place continuously for several hours or even days until the general condition of the patient has improved. A tourniquet which has been in place for one to several days must never be removed or allowed to slip; otherwise rapid and fatal intoxication may ensue. When operation is feasible, a second tourniquet is applied proximal to the first, which is then removed.

REFERENCES

- Adriani, John: *The Pharmacology of Anesthetic Drugs*, ed. 2, Springfield, Ill., 1941, Charles C Thomas.
- Barach, Alvin L.: *Physiologic Therapy in Respiratory Diseases*, ed. 2, Philadelphia, 1948, J. B. Lippincott Company.
- Clement, F. W.: *Nitrous Oxide-Oxygen Anesthesia*, Philadelphia, 1939, Lea & Febiger.
- Flagg, Paluel J.: *The Art of Anesthesia*, ed. 7, Philadelphia, 1944, J. B. Lippincott Company.
- Gillespie, Noel A.: *Endotracheal Anaesthesia*, Madison, Wis., 1941, University of Wisconsin Press.
- Guedel, Arthur E.: *Inhalation Anesthesia*, New York, 1937, The Macmillan Company.
- Hewer, C. Langton: *Recent Advances in Anesthesia and Analgesia*, ed. 4, Philadelphia, 1943, The Blakiston Company.
- Keys, Thomas E.: *The History of Surgical Anesthesia*, New York, 1945, Schuman's.
- Labat, Gaston: *Regional Anesthesia*, ed. 2, Philadelphia, 1928, W. B. Saunders Company.
- Leigh, M. Digby, and Belton, M. Kathleen: *Pediatric Anesthesia*, New York, 1948, The Macmillan Company.
- Lull, Clifford B., and Hingston, Robert A.: *Control of Pain in Childbirth*, Philadelphia, 1944, J. B. Lippincott Company.
- Macintosh, R. R., and Pratt-Bannister, F. B.: *Essentials of General Anaesthesia*, Oxford, 1943, Blackwell.
- Maxson, Louis H.: *Spinal Anesthesia*, Philadelphia, 1938, J. B. Lippincott Company.
- National Research Council, Division of Medical Sciences: *Fundamentals of Anesthesia: An Outline*. By Sub-committee on Anesthesia of Division of Medical Sciences, ed. 2, Chicago, 1944, American Medical Association.
- Southworth, James L., and Hingston, Robert A., editors: *Conduction Anesthesia: Clinical Studies of George P. Pitkin*, Philadelphia, 1946, J. B. Lippincott Company.

CHAPTER VIII

SURGICAL TECHNIQUE

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PRINCIPLES OF WOUND HEALING

Problems relative to the healing of wounds, acquired because of trauma or surgical intervention, are of fundamental importance. A surgeon's interest in and ability to cope with such problems are an accurate measure of his technical skill. Failure to appreciate the principles involved, which are few in number and entirely self-evident, produces disastrous results. An understanding of the natural forces of healing, of the sequence and timing of events in the process of repair and of factors which on the one hand expedite and on the other retard this process, is essential to good surgery.

Wounds are classified as:

- 1 Abrasions.
- 2 Contusions
- 3 Lacerations, with or without contusion, abrasion or loss of substance
- 4 Burns
- 5 Incised wounds, clean, contaminated or infected

The first two categories will not be considered at present. The third, concerning traumatic lacerations and associated complications, will be covered under wound excision and débridement later in this section.

The healing process in surgically incised wounds is precisely the same as that of traumatic lacerations, except that it is usually quicker, with less deformity and loss of function. The rate of healing and the ultimate anatomical and physiological results obtained depend to a very great extent upon the technique of the operator. Other contributing factors will be dealt with subsequently.

Surgical dissection may be *sharp* or *blunt*. Sharp dissection is carried out with the blade

of the knife; blunt dissection, with scissors, the handle of the knife, any suitable instrument, a gauze sponge, or the surgeon's finger. For the most part, sharp dissection is preferable. It effects clean exposure and creates relatively little trauma. Blunt dissection is employed as a minor complement to sharp dissection. Cleavage planes yield nicely to strokes of the blade together with an occasional gentle push by the handle of the knife. Forceful blunt dissection is never to be condoned. It tears and contuses the tissues, opens up potential dead spaces far beyond the required field and predisposes to hemorrhage and infection.

Control of hemorrhage is essential. A surgeon's efficiency may be judged by the dryness and neatness of his wound. It is impossible to operate effectively, and dangerous to attempt to do so, in the presence of uncontrolled bleeding. Whenever possible, vessels should be seen and accurately clamped with finely pointed hemostats before being cut. Together with the vessel, the clamp should crush little or none of the adjacent tissues. Ligation of large bites of tissue is to be deplored. Ligature by transfixion is recommended for all large vessels and for some small ones, when it is difficult to stabilize the knot. Transfixion also makes possible the use of more delicate suture material than would be required otherwise. Within reason, the fewer and less bulky the ligatures, the better the healing of the wound. Experience teaches which smaller vessels must be tied and which may be left to spontaneously contract and thrombose. Hematoma formation in a clean incision, besides delaying wound healing and predisposing to infection, indicates poor surgical technique.

Adequate reconstruction of a surgical incision implies accurate apposition of the various fascial planes without tension, obliteration of dead spaces and absolute hemostasis. The tensile strength of the sutures need be no greater than that of the tissues they are designed to secure. The sutures should take small bites and must not be tied so tightly as to cause necrosis of the tissues which they compress. Bulky suture material of any sort complicates wound healing. The less foreign material present in an incision the better. Each suture, therefore, should be cut as close to its knot as is compatible with security.

able to him. Operations are classified as clean, contaminated, and infected. Elective hernia repairs, mastectomies and thyroidec-tomies are examples of clean operations. Interval appendectomies and cholecystectomies may be minimally contaminated. Stomach and bowel resections are contaminated. Cases dealing with acute or subacute inflammatory lesions are said to be infected.

A clean wound may be repaired with either absorbable or nonabsorbable sutures. In a contaminated case, which becomes infected, nonabsorbable sutures predispose to sinus formation, except in situations where they are rapidly sealed off as occurs in the

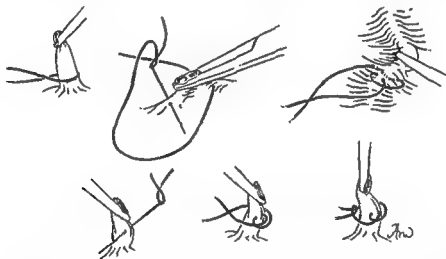


Fig. 28—Methods of vascular ligation. The first diagram represents the common manner of simple ligation. The others show various stages and types of ligation by transfixion. These last have been modified from those of Halsted which appear in his monograph on "The Use of Silk in Surgery," published by the Welch Bibliophilic Society in 1939.

Suture materials are classified as *absorbable* or *nonabsorbable*. The former group includes various types of catgut, plain or chromicized; the latter, such materials as silk, cotton, stainless steel, silver and numerous plastics. They are available in various sizes, from the extremely fine to the very coarse.

The choice of suture material is governed by the nature of the operation and the presence of contamination or infection. It is also influenced by the individual preference of the surgeon and by the facilities which are avail-

able to him. Operations are classified as clean, contaminated, and infected. Elective hernia repairs, mastectomies and thyroidec-tomies are examples of clean operations. Interval appendectomies and cholecystectomies may be minimally contaminated. Stomach and bowel resections are contaminated. Cases dealing with acute or subacute inflammatory lesions are said to be infected.

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led Halsted to adopt silk. His meticulous personal use of this medium and his instruction of others soon developed, at the Johns Hopkins Hospital, a school of surgery which, on this continent, remained for many years unique. George Heuer's monograph on Halsted's life and work, which was published in the *Bulletin of the Johns Hopkins Hospital* (February, 1952) on the occasion of the centennial of the birth of this great teacher surgeon contains (page 80) the comment which it is felt should be here quoted to inform the student of the qualities of the man whose influence on North American surgery may never be surpassed. "During his Baltimore period he was the exemplification of the slow, careful, painstaking surgeon who is concerned with the patient's safety, not with his own brilliancy. He was aware then, as most of us now are, that safe and successful surgery is the result, not of a rapid and dexterously performed operation, but of a combination of factors which includes the routine preliminary preparation of the patient, selected and well-administered anesthesia, perfect aseptic surgical technique, the gentle handling of all tissues exposed, the careful avoidance of injury to all structures not involved in the operative procedure, and the use of fine delicate instruments and suture material of fine silk in the control of hemorrhage and the closure of the wound. No one appreciated more keenly than he that the safety of the patient and the success of an operation can be jeopardized by a faulty anesthetic, by infection, by injury to an important structure, or by failure of proper wound healing."

Yet silk has never received general acclaim, despite the experimental and clinical evidence in its favor provided by many distinguished surgeons since Halsted's time. This is not difficult to explain. Silk demands the utmost in aseptic technique, in painstaking surgical judgment and skill. The requirements of ideal wound healing must be appreciated and fulfilled with precision; minimal trauma, rigorous hemostasis and

anatomical reconstruction without tension. As Halsted remarked, "In the hand of a bad technician silk is disastrous."

While there is no doubt that, under identical conditions, the healing process in a "silk" wound exhibits considerably less inflammatory reaction than in a "catgut" wound, the gross difference is not nearly so marked when catgut of comparable delicacy is used with the same precautions against trauma, tension, and hemorrhage as are required by silk.

The work of Howes and others has dissipated the illusion of catgut allergy which was formerly blamed for the occasional disruption of wounds repaired with absorbable sutures. If the contributory factors of tissue devitalization and increased tension are present in sufficient degree, a wound will disrupt as readily through sutures of catgut, silk or steel. It seems reasonable to conclude that a careful and well-trained surgeon, working under suitable conditions, will use silk (or other equally inert and delicate material) in clean wounds; but that, when in any doubt regarding the presence of contamination or infection, he will rely on fine catgut handled in the same manner.

WOUND EXCISION

Wound excision consists in the systematic removal of all devitalized or grossly contaminated tissue from a recently inflicted wound. It attempts to convert a contaminated and potentially infected lesion into one that is relatively clean. Depending upon the nature and site of the wound and the degree of its contamination, the time factor is somewhat elastic. Early, adequate excision of a contaminated wound is usually followed by uncomplicated healing. Wound excision following bacterial proliferation is dangerous because it removes natural barriers to spreading infection. Generally speaking, it is safe to perform wound excision within twelve hours of injury. In selected cases, minimally contaminated and presenting no sign of infection, the safe period may be extended a few hours.

The excision of a wound is performed in layers from without inward. Dead spaces are eliminated and hemostasis secured. Adequate drainage is established. The wound is enlarged, depending upon the extent of its penetration, by suitably placed incisions, which may increase its greatest diameter four to six times. Contused skin edges are cut away with a scalpel. As little healthy skin as possible is sacrificed in this procedure. Subcutaneous fat that is flecked with foreign material, severely contused or infiltrated with blood, is then removed. Deep fascial planes, which might prevent adequate drainage, are incised, preferably parallel to their fibers, but, when necessary, transversely. The viability of traumatized muscle is determined by its ability to contract when compressed by forceps. When in doubt, it is safer to excise more than less, for ischemic muscle is an ideal medium for gas gangrene infection. In compound fractures, only those bone fragments which are completely separated from periosteal and muscle attachments should be removed.

When practical, it is advisable to change gloves and dissection instruments after the skin layer has been excised. Some advocate voluminous saline irrigation of the wound with each succeeding step of excision. We feel that irrigation may be at times helpful, but that, in excess, it obscures the extent of contamination and may, in fact, spread potentially infected material beyond the natural confines of the wound. We prefer not to irrigate wounds that are excisable.

The excised wound is lightly filled, not packed, with dry, fluffed dressing gauze, after its walls have been covered with a single layer of finely meshed (bandage) gauze or silk, of which the interstices are small enough to prevent the inward growth of granulation tissue. This dressing is secured by an elastic bandage, and the lesion is immobilized by splintage or a plaster cast.

In excisable wounds, we feel that topical chemotherapy, applied as a thin film of finely particulate powder, is indicated. A

mixture of the more soluble sulfonamides and streptomycin is recommended for this purpose.

The term *débridement*, as applied to wounds, is by some considered synonymous with excision. We prefer, however, to confine its use to describe the management of wounds which are unsuitable for excision. In such cases, the lapse of time between injury and operation is commonly much longer than the safe period for excision. There is gross tissue devitalization, and suppuration is evident. Infection is established to a moderate or marked degree.

The aims of *débridement* are to relieve tension, to remove such necrotic tissue and foreign material as is readily accessible, without disrupting natural barriers to spreading infection, and to effect adequate drainage of all regions of the wound. Tension is diminished by incision of confining fascial planes. Loosely adherent slough is clipped away. Foreign bodies are removed when this may be accomplished without extensive exploration. Dead spaces are unroofed and the defect is filled with dry, fluffed gauze dressings. Counterdrainage is established when indicated. It is doubtful that topical chemotherapy of any sort is effective in such wounds, or that their irrigation with antiseptic solutions speeds the healing process. Most important is their complete immobilization, preferably in highly absorbent plaster of Paris casts. Infrequent dressings are definitely indicated. Each change of dressing inflicts trauma, delays healing and increases the incidence of cross infection. These facts were established by Trueta during the Spanish Civil War and have stood the test of time since then.

CLOSURE OF ACCIDENTAL WOUNDS

Small, superficial, cleanly incised wounds, within twelve hours of infliction and following excision of grossly contaminated fat, fascia, and devitalized skin, may be closed safely by *primary suture*. Larger, more

deeply penetrating wounds, though recent, are better treated by excision and closed by *delayed, primary suture*, after 48 to 72 hours, or by *secondary suture*, after 7 to 14 days or longer. In such cases, the optimum time for closure is when the entire defect is lined by healthy granulation tissue, when no dead space remains, and when all slough has separated.

Wounds that, for the reasons mentioned, require débridement rather than excision may be closed by secondary suture after an interval of several weeks. Because of excessive loss of substance, however, they often require one or other type of definitive plastic repair.

SUPERVISION OF WOUND HEALING

The incidence of wound healing complications is inversely proportional to the surgeon's technical ability and the efficiency of his operating team. It is obvious, therefore, that such complications should be scrupulously recorded and subjected to frequent review. By means of this information, errors in surgical judgment and technique, in sterilization procedures and so forth, are detected and eliminated without delay. Infection, nutritional deficiencies, metabolic disorders, blood dyscrasias and numerous other factors impede the normal process of repair, to such an extent that the more serious complications of wound healing are, at times, inevitable. To record only such major catastrophes is of little value. The picture must be complete—either a surgical incision heals cleanly by primary union or it does not. If the slightest complication occurs, it should be noted in the progress report and discharge summary of the case.

To properly assess the quality of wound healing, the type of case—clean, contaminated or infected—must be stated, suture materials and drainage devices noted, and healing summarized as follows: (a) healed cleanly by primary union; (b) stitch abscess;

(c) hematoma; (d) trivial infection; (e) serious infection; (f) wound disruption.

An accurate statistical record of wound healing, presented as an annual report, will clearly demonstrate technical improvement or the reverse.

SURGICAL STERILIZATION

Surgical sterilization implies absolute destruction of bacteria. It is accomplished by physical or chemical processes: by immersion in boiling water for at least 10 minutes; by autoclaving at 18 pounds pressure (254° F) for varying periods depending upon the article to be sterilized; by dry heat in the hot air oven, or by chemical reagents, such as ethyl alcohol (70 %), oxycyanide of mercury (1:1,000) biniodide of mercury or numerous other antiseptics.

Routine procedures are devised for the maintenance and sterilization of all types of surgical equipment. A few of the commoner methods should be mentioned. Packaged dry goods, granite ware, rubber goods (including drainage material), silk sutures, brushes and glassware are autoclaved for 30 minutes; rubber gloves for 15 minutes; and instruments for from 10 to 20 minutes. Articles that may be damaged by intense heat are immersed in appropriate antiseptic solutions for varying periods.

SCRUBBING FOR OPERATIONS

It is impossible completely to sterilize the hands of the operating team. Numerous reagents, because of their bactericidal and detergent properties, reduce bacterial flora to a minimum, but surgical sterilization is never accomplished. Systematic scrubbing for ten minutes with a bland, superfatted soap and brush of not too coarse texture, followed by one minute's immersion of the rinsed hands and forearms in a trough of mild antiseptic (aqueous Zephiran, 1:1,000) is an effective routine. Frequent scrubbing facilitates cleansing. Adjuvant detergents and

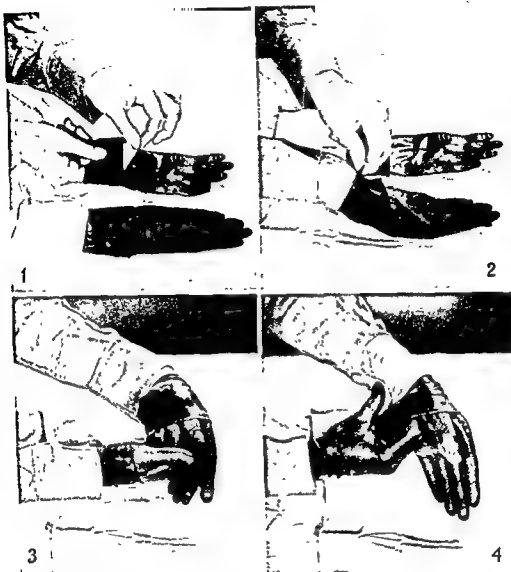


Fig. 29—Application of rubber gloves.

1. Method of removing powder package from glove cuff.
- 2 and 3. Method of putting on gloves; hand-to-hand and glove-to-glove technique.
4. Method of rolling glove cuff over cuff of gown

antiseptics may reduce scrubbing time to as little as three minutes.

The following points concerning scrub-up must be emphasized:

1. There must not be any infection of the skin or break in skin continuity (abrasion or laceration) of the hands or forearms.
2. The nails should be clean, smooth and pared short
3. The scrub-up zone should include the elbows.

PREPARATION OF THE OPERATIVE FIELD

The night before operation, the area is cleansed with green soap and carefully shaved far beyond the limits of incision. In the operating room further cleansing is carried out with alcohol and ether, and an antiseptic solution is applied in systematic strokes from the center of the field to its periphery. Contaminated zones are painted last.

For orthopedic operations it is customary to supplement the above procedure by one or more similar treatments on the ward during a period of 24 to 48 hours prior to operation. The limb is wrapped in sterile towels following each treatment. In such cases of repeated application it is particularly important that the antiseptic used should be nonirritating.

should avoid compromising major nerves and blood vessels and, when practical, divide fascial aponeuroses in the direction of their fibers rather than transversely. Except in cases of emergency, the speed with which an incision may be opened and closed is far less important than that it should fulfill the above requirements insofar as is reasonably possible.



Fig. 30—Commoner breaks in aseptic technique: 1 Second assistant's left arm outside sterile field 2 Inadequately draped anesthesia screen 3 First assistant improperly masked 4. Instrument nurse's hair uncovered by cap 5 First assistant reaching behind instrument nurse 6 Hustle nurse's uniform touching sterile hand basin 7 Surgeon's hand below level of sterile field 8 Surgeon's cuff outside glove 9 Second assistant's retractor outside sterile field.

SURGICAL INCISIONS

An incision is designed primarily to afford the best possible exposure of the lesion in question, with minimal retraction of adjacent structures. It should, for cosmetic reasons, parallel the natural creases of the skin. Its course should be at a right angle to the minimal distracting forces in the area. It

The closure of an incision is often a tedious procedure, but the time consumed in meticulous reconstruction is never wasted. Accurate approximation of serosal surfaces, fascial planes and skin edges, with delicate sutures under minimal tension, rigorous hemostasis and obliteration of dead spaces are basic to adequate surgery.

DRAINAGE OF WOUNDS

In clean operations drainage is seldom required and should be avoided, because the drain itself creates a dead space, acts as a foreign body, and affords access to infection from without. In many cases, drainage of clean wounds indicates indolence or ineptness on the part of the surgeon, who has failed to effect hemostasis. Occasionally, when oozing is difficult to control or when removal of a large tumor leaves a dead space which may fill with serosanguineous fluid, drainage for a period of 24 to 48 hours is advisable.

2. Residual necrotic tissue.
3. Bleeding such as is controllable only by packing.
4. Operations involving the gall bladder, bile ducts or liver parenchyma.
5. Unsatisfactory inversion of the duodenal stump.
6. Operations on the pancreas.
7. Grossly contaminated colon anastomoses.

Drainage of the abdomen in the presence of acute, diffuse peritonitis is ineffectual, because whatever device is used will be walled off from the major cavity within a few hours.

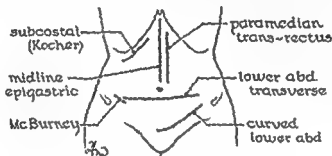


Fig. 31.—Sites of the more common abdominal incisions.

Whether or not to drain a contaminated wound is decided by the amount and character of the contaminant and the natural resistance of the tissues immediately involved. When in doubt, it is safer to drain such wounds until it is apparent that no infection has taken place.

Infected wounds must be left open and drainage maintained by tamponade or tube. If precautions are taken to protect the subcutaneous fat from contact with deeply seated infected material, it is usually safe to close the angles of the wound up to the drainage tract and thereby shorten the time of healing.

Indications for drainage of abdominal incisions merit special consideration. Normal peritoneum is amazingly resistant to infection. Once inflamed, however, it rapidly loses this happy faculty. The following conditions necessitate drainage of the peritoneal cavity:

1. Localized abscess.

Though the peritoneum may be closed safely in many cases of contamination or infection, it is advisable, by suitable drainage, to anticipate involvement of the less resistant extraperitoneal areolar tissue and subcutaneous fat. Delayed primary closure of the skin is an effective safeguard when nonabsorbable sutures are used to reconstruct a potentially contaminated incision.

INSTRUMENTS

In many teaching hospitals, surgical internes serve for several months as "instrument nurses," becoming thoroughly familiar with the proper names and applications of the tools of their trade. Though this luxury is impractical for the student, and the consensus that details of surgical technique should not complicate undergraduate teaching is probably correct, we feel that the student should be encouraged to learn the names and purposes of instruments in common use. The accompanying photographs

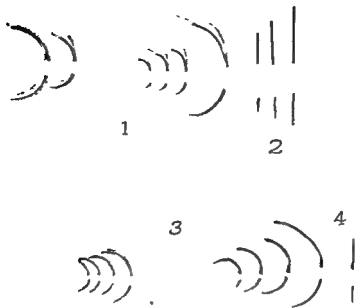


Fig. 32.—Needles 1 Heavy fistula and surgeon's cutting edge needles 2 Keith's abdominal needles 3 Set of Ferguson and Mayo round-bodied, curved needles 4, Straight, round needles (FGH).

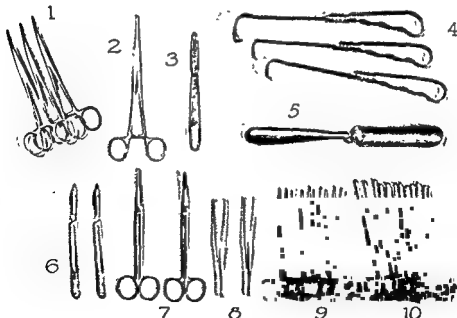


Fig. 33.—Basic dissection instruments 1 Curved Pean forceps 2 Ochsner forceps 3 Kocher dissector. 4 Richardson retractors. 5 Abdominal spoon. 6 Bard-Parker blades and handles 7 Mayo scissors, straight and curved. 8 Toothed and plain tissue forceps. 9 Straight artery forceps 10 Allis forceps

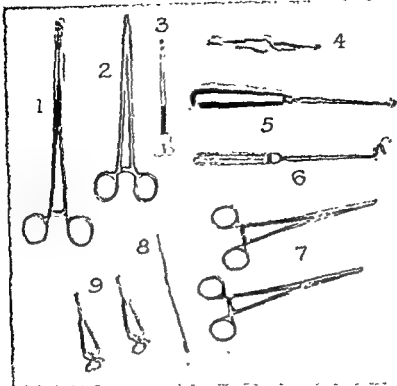


Fig 34—Instruments supplementary to basic dissection. 1. Foerster sponge forceps. 2 Mayo needle holder 3 Grooved director. 4. Hagenbarth-Michel clip holder. 5 Curette. 6 McEwen needle holder. 7 Ochsner clamps 8 Probe. 9. Jones towel clip.

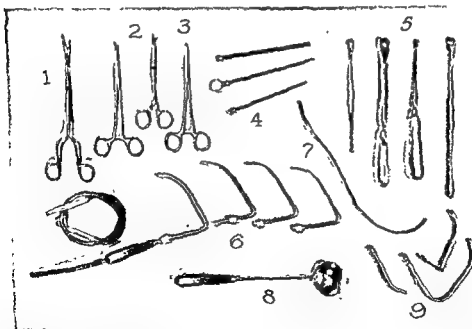


Fig 35—Gall bladder and common duct instruments 1. Common duct stone forceps 2. Lahey forceps and right-angled duct forceps. 3. Artery forceps (7½"). 4. Set of suction tips with universal handle (R.V.H.). 5 Gallstone scoops and curette. 6 Common duct probes and irrigators (Archibald) with universal handle (R.V.H) 7. Common duct metal catheter. 8. Moore gall bladder spoon 9 Common duct suction and T-tube inserter (R.V.H)

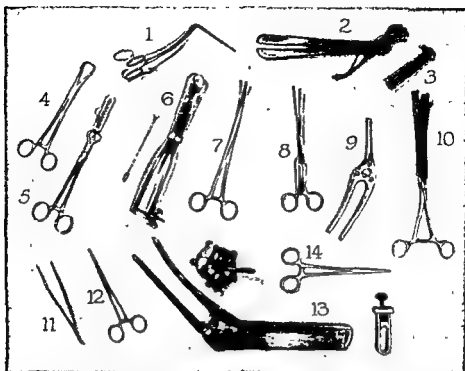


Fig 36—Stomach and bowel instruments 1 Right-angled bayonet forceps 2 De Martel clamp. 3 Set of blades for De Martel clamp 4. Hendrickson clamp 5 McClure clamp 6 Furniss clamp and special needle for intestinal anastomosis 7. Curved Pean forceps 8 Bayonet forceps 9 Payr's clamp 10 Rubber covered intestinal forceps (Kocher) 11 Leedham-Green forceps 12. Miller-Allis forceps 13. Von Petz clamp for stomach and intestine 14 Artery forceps (7 1/2").

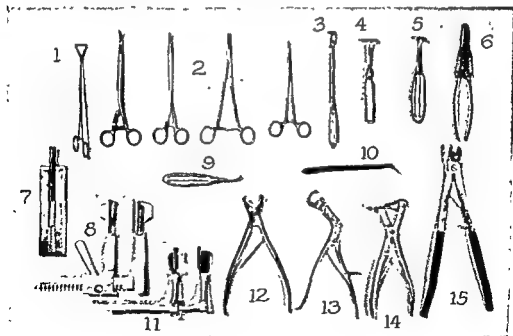


Fig 37—Chest instruments 1 (a) Duval-Grille lung grasping forceps, (b) Moynihan forceps; (c) Gray forceps 2 (a) MacKenzie silver clip holder; (b) Lahey forceps. 3. Right-angled raspatory and elevator 4 Doyen costal elevator 5 Bethune elevator. 6 Double-action rongeur (Barton). 7. MacKenzie silver clip magazine 8. Finochietto retractor 9. Goose-neck raspatory. 10 Right-angled Bard-Parker blade handle 11 Tuffier rib retractor 12. Right-angled rib cutter. 13 Giertz-Shumaker rib shears (Stille). 14. Shumaker rib shears.

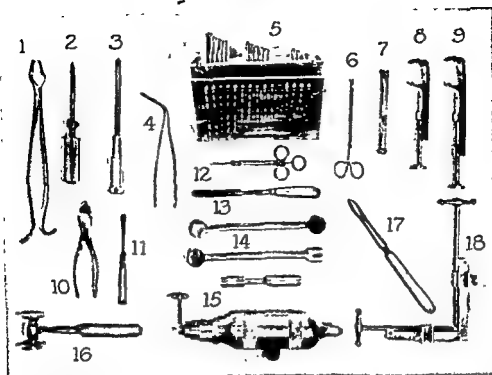


Fig. 38—Fracture instruments. 1. Lane bone-holding forceps. 2. Screw-driver (Zimmer). 3. Screw holder and driver (Down Bros). 4. Plate holding forceps. 5. Set of Vitallium plates and screws. 6. Screw-holding forceps. 7. Osteotome. 8, 9 and 18. Set of Lowman bone-holding forceps. 10. Wire cutting forceps. 11. Lane periosteal elevator. 12. Depth finder (Down Bros). 13. Bone file. 14. Zimmer plate-bending forceps. 15. Wire twister (Down Bros.) and electric motor for drills and circular saws (Luc.). 16. Metal mallet. 17. Lane elevator and metal rule.

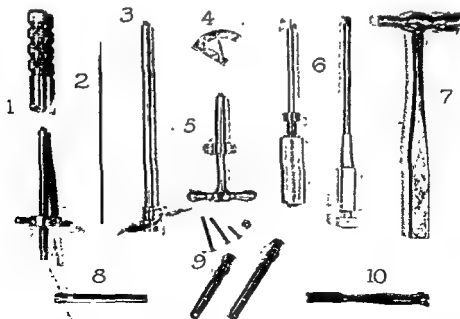


Fig. 39—Hip pinning—Smith Petersen instruments. 1. Nail extractor. 2. Guide wire. 3. Nail inserter. 4. Protractor. 5. Pin extractor. 6. Screw driver and screw holder (Down Bros.). 7. Metal mallet. 8. Nail. 9. Side-plates and screws. 10. Nail starter.

display instrument setups routinely associated with various operative procedures at the Royal Victoria Hospital

SUTURES

Methods of wound reconstruction are governed by many factors. The site of incision, the nature of the lesion, the nutritional state of the patient, the possibility of contamination or infection, and the requirements of drainage all must be considered in the evolution of a suitably balanced technique.

Continuous sutures should be avoided, except in the mucosal layer of intestinal anas-

inversion of the apposed edges. Plain, interrupted stitches suffice for flat or convex skin surfaces and most fascial planes. In concavities, such as the groin, vertical mattress sutures are essential to prevent inversion of the skin. The far-and-near interrupted suture, recommended by Whipple for appropriate fascial planes, accomplishes accurate approximation without tissue strangulation. In debilitated subjects the Jones vertical figure-of-eight interrupted suture, which includes all layers of the abdominal wall except subcutaneous fat and skin, is most effective in closing vertical paramedian incisions. When properly tied, these stitches

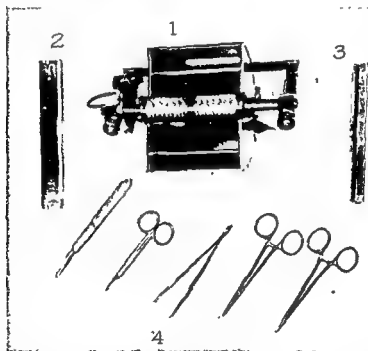


Fig. 40—Skin grafting instruments 1 Paget dermatome 2 Blade for dermatome 3 Blade holder 4 Bard-Parker blade handle, plastic scissors (Stille), Adson forceps, curved mosquito forceps and straight mosquito forceps.

tomosis and the closure of peritoneum or synovia. A continuous, removable, subcuticular stitch is useful in some cases. Interrupted sutures are described as plain, mattress or figure-of-eight. Numerous additional modifications need not complicate this text. Mattress sutures more securely grip the tissues they approximate and can be placed so as to effect either eversion or

create a smooth peritoneal surface and do not tear out. The small loop through the anterior sheath of the rectus helps to prevent excessive constriction of the larger bite of peritoneum, muscle and fascia.

SURGICAL DRESSINGS

Clean wounds, when not drained, require no more than a few layers of plain gauze,

secured by short strips of adhesive. In the presence of drainage, a small absorbent pad is added.

When pressure is required, to obliterate dead spaces or prevent oozing, fluffed gauze and cotton waste are spread evenly over the innermost dressing and its environment and compressed by elastic adhesive tape.

The dressing of accidental wounds is dealt with in the section on excision and débridement.

BANDAGES

The art of bandaging, so meticulously fostered by the French school of a century ago, is lost to many contemporary surgeons. As a rule, the student and interne receive

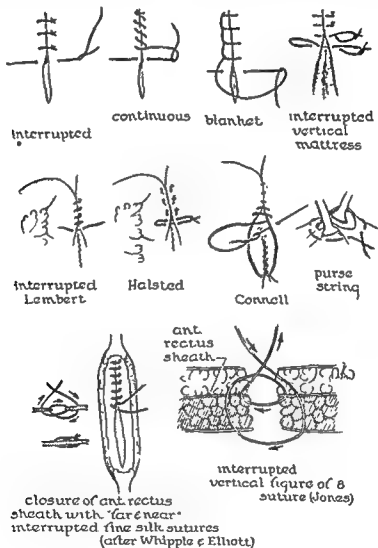


Fig 41—Various types of sutures.

To prevent it from slipping beneath the surface of the skin, the end of a drainage tube must be transfixed by a safety pin. If drainage is intended to be of brief duration, a thread, attached to the safety pin and brought out at the margin of the dressing, permits withdrawal of the drain without exposure of the incision.

most of their practical instruction in bandaging from nurses in charge of out-patient clinics, whose technique is usually excellent. Adhesive tape has, to a great extent, replaced the bandage.

Bandages are designed to fit conical or cylindrical surfaces, or a combination of these requiring figure-of-eight turns, and re-

ferred to as a spica bandage. If the surface is cylindrical, spiral convolutions with uniform (2/3) overlapping are sufficient. If the region is conical, a spiral bandage is started at the narrower extremity and reversed when necessary to accommodate the increasing diameter of the part. A spica bandage, secured distally and proximally by two circular turns, consists of a series of overlapping figure-of-eight convolutions and affords best support to dressings at the junction of one or other extremity with the trunk.

Bandages which envelop dry dressings are best maintained by spiral strips of adhesive tape. When moist dressings are prescribed, commonly in minor infections of fingers or toes, the bandage is first made fast by two or

is infinitely preferable to the rigidity of plain gauze. Inelastic adhesive strapping must never encircle any part of an extremity, nor may circular bandages of any rigid material be applied beneath plaster casts.

PLASTER CASTS

Plaster of Paris is still the medium of choice for immobilizing injured tissues. It is rubbed into strips of coarse muslin which are then rolled into bandages or folded into slabs of various lengths and widths. The bandage or slab, having been dipped in tepid water and drained of excess fluid, is ready for use. In hospital and office practice, proprietary plaster bandages and slabs have largely replaced the homemade variety.

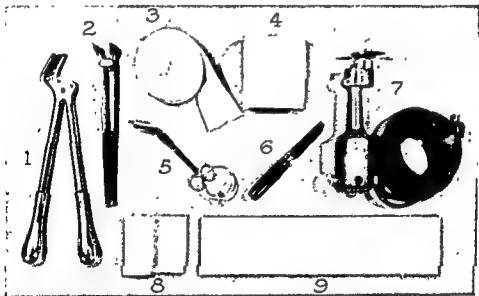


Fig 42—Plaster of Paris cast instruments 1 Stille-type plaster of Paris spreader 2 Cast spreader 3 Stockinette 4 Sheet wadding 5 Plaster scissors (Lister bandage). 6 Plaster knife. 7 Stryker cast cutter 8 Plaster bandage 9 Plaster slab

three turns at wrist or ankle, carried obliquely across the dorsum of hand or foot to secure the dressing of the affected digit and then back again in the opposite oblique to be tied at its proximal origin.

Bandages should afford uniform, firm support to dressings. They must never be so tight as to impede circulation or exert excess pressure on bony prominences or superficial nerves. The resiliency of an elastic bandage

Their neatness, delicate texture, uniform impregnation, and rapid setting are distinct advantages, which more than compensate for diminished absorbability.

The requirements of a plaster cast are accurate coaptation in no greater thickness than the situation demands, and complete immobilization of the part, by inclusion of joints proximal and distal to the lesion, in a position of optimal function. When open

wounds are thus immobilized, the absorptivity of the plaster is of great importance. If this quality is impaired, tissue fluid accumulates beneath the cast with consequent maceration of skin, edema of the granulating surface and inevitable infection. The stain of serosanguineous drainage should appear on the surface of a suitably absorbent cast within a few hours of its application.

Strategic use of plaster slabs greatly diminishes the bulk of a cast without lessening its mechanical efficiency. The slab, of any required thickness, is moulded to the under (weight-bearing) surface of the extremity and secured by a circular bandage of which 2 to 4 layers generally suffice. Such a cast is easily removed by a longitudinal cut through its thinner surface and eversion of the cut edges. A thick, cumbersome, irregular cast demonstrates the inexperience of the operator. If the individual who applies a cast is also responsible for its removal, he will soon take steps to improve his technique.

When applied during the acute phase of injury, plaster casts must be carefully padded to accommodate increased swelling and avoid pressure on traumatized soft tissues. For this purpose, a few layers of sheet wadding are effective. Bony prominences, superficial nerves, and weight-bearing areas are additionally protected by pads of felt. At this stage, continual elevation lessens edema and improves circulation. If constant supervision of the case is impossible, as when battle casualties are evacuated from forward areas, it is customary to split the freshly applied cast longitudinally through its thinner surface. This is accomplished most simply when a flattened roll of paper (newsprint is satisfactory), comprising several layers about 2 cm. wide, is placed for protection of the skin in the long axis of the limb opposite the slab and incorporated in the circular bandage. While the plaster is still soft, it is easily incised by a sharp blade down to the protective layer of paper. When the split is complete, its edges will spring

several millimeters apart, but further separation is prevented and the function of the cast fully maintained by two or three circular strips of gauze bandage. If, during transportation of the case, swelling should obstruct circulation or infection necessitate further treatment of the wound, the properly split cast may be opened in a matter of seconds by the fingers alone.

When the acute reaction to injury has subsided, muscle atrophy and decreased swelling require that the cast be changed for one that snugly fits the contours of the shrunken limb. The new cast needs relatively little padding. In simple fractures, it is moulded over stockingette plus a turn or two of sheet wadding in pressure areas. In compound injuries with persistent drainage, it is applied preferably over sheet wadding which, however, should not be interposed between the absorbent wound dressing and the cast.

The quality of a cast is directly proportional to the operator's experience and personal interest in its composition. Plaster technique, like formal sculpture, is not acquired overnight. Numerous devices in padding, smoothing and moulding greatly lessen the patient's discomfort and expedite restoration of function. Finally, it is axiomatic that when a patient complains of localized pressure beneath a cast he is always right until the affected area has been unroofed for inspection. The pressure lesions caused by ill-fitting casts are often more serious than the initial injury.

REFERENCES

- Halsted, William S.: The Employment of Fine Silk in Preference to Catgut and the Advantages of Transfixing Tissues and Vessels in Controlling Hemorrhage, *J. A. M. A.* 60: 1119-1126, 1913.
- Heuer, G. W.: Dr. Halsted Bull Johns Hopkins Hosp. (Supplement) 90: p 80, No. 2, Feb., 1952.
- Howes, E. L.: A Renaissance of Suture Technique Needed, *Am J Surg.* 48: 548-552, 1940.
- Whipple, A. O., and Elliott, R. H. E., Jr.: The Repair of Abdominal Incisions, *Ann. Surg.* 108: 741-751, 1938.

CHAPTER IX

PLASTIC SURGERY

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GENERAL CONSIDERATIONS

Ancient records of the Egyptians and Indians, written many centuries ago, reveal that they had developed methods of repairing mutilated and deformed features, thus, the origin of this specialty is deeply rooted in the past. Great advances, however, have been achieved in this branch of surgery since the beginning of the twentieth century. This revival of interest has been stimulated by World Wars I and II, the invention of new instruments, and recently discovered therapeutic agents.

Plastic surgery deals with the repair of congenital or acquired defects with a view to restoration of function, improvement of appearance and resolution of any psychological disturbances caused by the defect. In recent years, more emphasis has been placed upon the latter aspect, because of the increasing number of handicapped persons injured by warfare and civilian accidents. A facial injury or malformation should be corrected early when possible to prevent the development of deep psychological wounds. If only partial or no improvement can be accomplished by surgery, psychotherapy should be instituted to aid the individual to become adjusted to the deformity.

Plastic surgery extends into some aspects of general surgery and various surgical specialties. Collaboration is often required between other specialists and the plastic surgeon. In many cases the special experience of the latter in planning incisions, designing flaps, and obtaining free grafts of various tissues contributes greatly to successful results. This is particularly true, for example, when extensive trauma of skin and subcutaneous tissues has occurred.

A basic sense of artistic imagination is required in which the various stages in their proper sequence, of a given reconstructive problem can be visualized. The most suitable method of treatment of a specific condition must be selected, due consideration having been given to the physical condition and status of the patient. It is important to diagnose whether tissues have been lost or only misplaced and how they may be restored. The general principles of wound healing and of the rate of healing of different tissues must be known. Gentle handling of tissues, employing sharp knives, scissors, skin hooks, and atraumatic needles with finest suture materials will aid healing. The blood supply of flaps or tissues being operated upon should be carefully preserved or the best planned operation will fail. Positive hemostasis should be obtained by fine ligatures since the use of pressure alone or hot towels may permit hematoma formation which may mar what might have been an excellent result. The type of dressing and amount of pressure applied vary with different procedures and should be considered as the last stage of the operation. The dressing should immobilize the tissues, approximate raw surfaces, minimize edema and oozing of blood or plasma but not interfere with adequate blood supply to the area. When elective plastic operations are being performed there should be no local pustules or general infective process present.

SKIN GRAFTS

A free skin graft may be defined as a portion of skin which has been completely severed from its position and has been transferred to another area of the body. The technique of skin grafting has been known

for centuries, and it was not an uncommon procedure for gluteal skin to be grafted in order to restore nasal defects. Through the years, surgeons such as Reverdin, Thiersch, Wolfe, Blair and others have described various types of skin grafts and methods of obtaining them. The methods of obtaining split skin grafts were revolutionized by Padgett in 1939 when he introduced the dermatome. Three main types of skin grafts are used.

Small Deep Skin Grafts.—These small fragments of skin vary in size from 4 to 8 mm in diameter and find their only use in areas which are grossly infected, where the blood supply is inadequate, and in lining chronic sinuses or cavities with overhanging walls. On the other hand, they produce certain disadvantages in that the donor area heals as an ugly flagstone-like, patchy scar which not only tends to cause keloid formation but renders the donor area unfit for supplying further grafts.

Technique.—Preferably under local anesthesia, or general if necessary, a straight needle, inserted in a wooden handle or hemostat, is used to pick up a small cone of skin, the base of which is cut through with a sharp scalpel, thus obtaining a small, hemispherical piece of skin which contains almost the entire thickness of the dermis in the center and tapers to a feather edge at the periphery. These grafts are placed on the granulating area, at intervals, and are then covered with a piece of paraffined-net which is cemented to the skin surrounding the defect with collodion or liquid adhesive. Subsequently, fluffed, gauze dressings moistened with saline are applied every few hours for 4 or 5 days, after which the net may be removed, revealing the already adherent grafts.

Full-thickness Grafts.—These are dissected from an area of the body which provides skin of suitable color, thickness and hair distribution to that of the recipient area. It possesses the advantage of having a good cosmetic appearance, contracting little, and

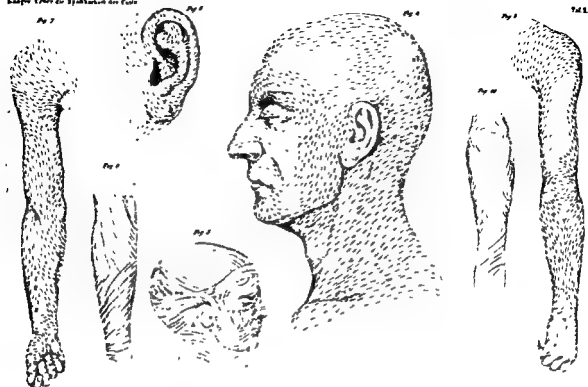
provides good weight-bearing and friction-tolerating surfaces. This type of graft is now most frequently used about the face and weight-bearing areas of the body. The most obvious disadvantages are that they will not "take" on infected areas, they cannot be obtained in large quantities, and they require extremely careful dressing.

Technique.—A pattern of the defect to be covered is made with sterile Pliofilm, celluloid or thin lead sheet. This is then placed on the donor area and the outline traced with a scalpel. One margin of the graft is then raised and, using a small cylindrical object as a roller, the graft is held on tension, which facilitates removal, and is gradually dissected from its bed at the junction of the dermis and subcutaneous fat, none of which should remain on the graft. This donor site is then closed by undermining and suturing the margins of the wound, or, if this is not feasible, by the application of a split-skin graft.

Dermatome Skin Graft.—This type of graft may be taken by any of the commonly used types of dermatomes now on the market which permit the cutting of a graft in accurately calibrated thicknesses from thin to almost full-thickness grafts. The *thin* grafts which are commonly used vary from 0.010 to 0.014 inch in thickness and are particularly suitable for grafting in children where the skin is relatively thin, and in granulating wounds where the take of a thick graft would be doubtful due to the paucity of blood supply or heavy contamination with bacteria. On the other hand, the *thick* dermatome grafts, which commonly vary from 0.018 to 0.024 inch in thickness, provide, in considerable measure, the advantages of the full thickness graft without the necessity for covering or closing the large defect which results from the removal of a full thickness graft.

Technique.—When the dermatome has set to the required thickness of the graft to be cut, the drum of the dermatome and the skin of the donor area are painted with rubber

Körperlicher Zustand des Patienten



Körperlicher Zustand des Patienten

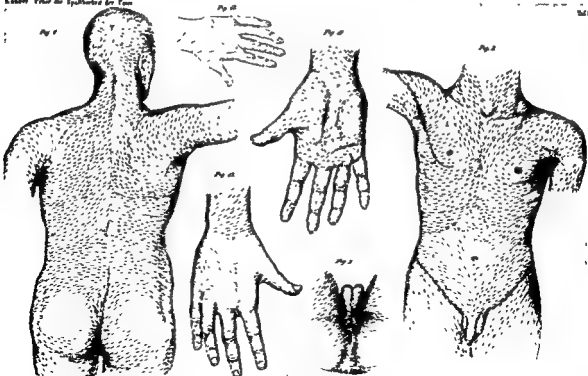


Fig. 43.—(See opposite page for legend.)

cement which is allowed to dry until the initial gloss of the cement has disappeared. The leading portion of the drum is then pressed firmly against the donor area, and when the adherence between the two is firm, the drum is rotated upward slightly and the knife handle is then slid backward and forward until the desired amount of skin is removed. At the end of the drum the skin may be severed with scissors or by pulling the drum away from the skin and cutting through with the dermatome blade.

Xeroform or petrolatum gauze and a firm pressure dressing which is changed at intervals of several days for a short period. When the dressing is changed, any necrotic tissue or exuberant granulations may be scraped away before applying the next dressing. Exudates should be washed away with saline or mild antiseptic solutions. When known pathogens are present, with an increased amount of purulent exudate, they may be controlled by the application of dressings, soaked in a solution of some effective



Fig. 43—Langer's conception of the lines of normal skin tension as determined by multiple puncture wounds with a round pointed metal awl in the skin of cadavers.

The common reasons for unsuccessful operations of the dermatome are a dull knife, cement that is too thick or too thin, moisture on the drum or on the skin. In emaciated patients saline may be injected into concavities such as between the ribs to aid in obtaining a complete drum of skin.

Granulating areas may be prepared for skin grafting by covering the region with

antibiotic for a day or two before the graft is applied. When the skin graft is applied to the raw area it may be laid on healthy granulations, or, if these are not healthy, they may be shaved off with a scalpel or sharp scissors, carefully, so that the tendons and other important structures are not exposed. The grafts are sutured in place with fine Dermalon and are dressed

with Xeroform fine mesh gauze, several layers of dressings followed by cotton waste and rubber sponge. The whole dressing is firmly secured with even, firm pressure by bandages and adhesive. Near-by joints should be immobilized by a padded splint.

Failure of skin grafts to live may be due to a variety of causes, both systemic and local. In the first category it may be noted that anemia is most detrimental to successful growth. It has been stated that a hemoglobin below 65% markedly decreases the chance of take of a skin graft. It is good practice not to perform an extensive skin

Skin grafts folded with the raw surface approximated and wrapped in moist gauze may be refrigerated at or slightly above 0° C for several weeks and then be transplanted successfully. If for some reason the patient's condition deteriorates, the operation may be terminated, and the remaining skin grafts preserved until a later date when the operative procedure may be completed with safety.

Autografts (self) are those taken from and applied to the same person and survive permanently. Grafts between identical twins

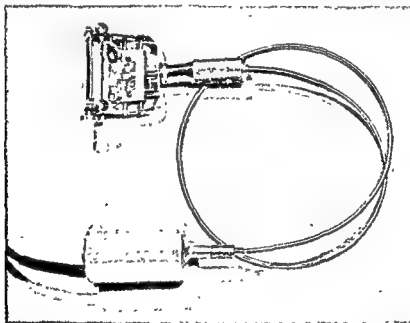


Fig 44—Dermatome. The electrodermatome may be used to obtain long skin grafts from any part of the body, and may also be employed to remove exuberant granulations.

graft if the hemoglobin is below 80 %. In patients with severe burns an effort is made to raise the hemoglobin to 90 % by transfusions before operating. Other general causes are a high postoperative fever, hypoproteinemia and hypovitaminosis. The most important local factors interfering with successful healing are an improper preparation of the granulating bed, the presence of virulent pathogenic organisms, insufficient or uneven pressure dressings, and inadequate fixation of joints.

may be included in this category since they are monozygotic. *Homografts* (same) are those between individuals other than identical twins. They survive for a few weeks and then slough. Homografts are useful as a temporary cover for extensively burned patients, while autografts are being applied in patchwork fashion between the homografts to obtain the maximum marginal growth. It may be necessary to obtain several crops from the same donor area to eke out the supply. *Heterografts* (other) or *zoografts*

are those from other species applied to man and are no longer used. The term iso-graft should not be used synonymously with homograft since iso means equal.

Corneal homografts can be performed successfully, possibly because this tissue is nourished only by lymph. Technical success in many cases was often reduced by clouding of the graft which gradually became opaque because of vascular ingrowth. The local application of aqueous suspension of cortisone to the eye postoperatively appears to be useful in reducing the incidence of cloudy grafts.

Mucous membrane grafts may be removed from the lip or cheek and be used to restore conjunctival lining of the eye or vermillion border of the lip. Skin grafts when used to replace conjunctiva produce an objectionable discharge.

Dermis.—Consists of the deeper elements of skin, the epithelium having been removed either with a dermatome or a razor. It may be inserted beneath the skin to fill depressions; it shrinks less than fat. Sheets of dermis have been used to reinforce large hernial defects. Thick dermatome skin grafts may be resplit on the dermatome by setting the blade at half the previous thickness and removing the deeper layer of the graft in a single sheet, thus doubling the surface area of the graft. The dermis takes well, and regenerates a new epithelium rapidly. The surface is paler than that of an ordinary skin graft because few pigment cells are present.

Fat may be used to fill depressions, particularly those about the face. Rigid asepsis and atraumatic technique are essential in obtaining the graft. Shrinkage after grafting amounts to about 40 % so that a defect should be overcorrected. It is less extensive if large pieces are grafted. Fat may be combined with dermis or fascia which provide a firmer tissue with less tendency to shrink due to either degeneration or absorption.

Fascia is most frequently used to form slings which are employed to support the drooping cheek and mouth following facial paralysis or to raise the eyelid in ptosis. Fascia is also used in arthroplasty, to replace tendons, or to prevent adhesions between tendons and adjacent structures. It is abundantly available from the fascia lata of the thigh from which it may be removed by direct exposure or through a small incision with a fascial stripper.

Muscle is rarely used as free grafts, but flaps of masseter or temporal muscle with blood and nerve supply preserved are employed to provide correction of facial paralysis.

Cartilage has many uses as a free graft, chiefly to fill in bony depressions about the face. It has many advantages since it does not shrink, may be carved easily, and provides a resilient support for soft tissue. Cartilage is probably most frequently used in the reconstruction of the saddle nose. Most surgeons prefer to use fresh autotransplants which do not absorb but either fresh or preserved homografts may be used, although marked absorption usually occurs in time.

Tendon grafts are almost exclusively employed to replace missing or damaged tendons. The palmaris longus tendon or the short extensor tendons of the foot, removed with paratenon, are those usually chosen for tendon grafting in the hand. Tendon grafts have been advocated instead of fascial slings in facial paralysis in the belief that fewer adhesions which limit motion would form about a tendon with its paratenon.

Bone grafts are indicated when a rigid supporting tissue is necessary and are used to repair mandibular, cranial, and sometimes malar and nasal defects. Pedicle grafts of bone and osteoperiosteal grafts are rarely used. Autografts are preferable in plastic surgery, although bone bank material may be used if circumstances contraindicate the use of autogenous bone. The usual source

is the crest of the ilium, but the ribs, tibia or fibula may be used. Cancellous bone chips from the ilium have been employed successfully in forming moulded bone grafts for reconstruction of contoural bone defects of the face. Two advantages of these grafts are that they can survive and consolidate even in the presence of mild infection, and that tedious carving is not required.

Composite Graft.—First described by Koenig, these grafts are obtained from the helix of the ear and consist of two covered surfaces and intervening cartilage. These may be used to repair defects of the ala, tip and columella of the nose, with excellent results. Full thickness loss of the eyelid has also been replaced with the use of two surface graft with a cartilage graft in between.

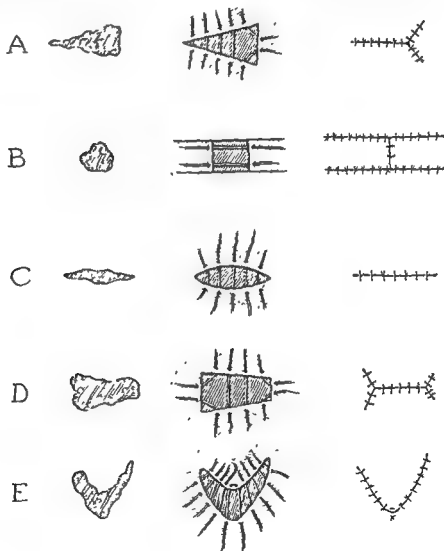


Fig 45—Various methods for excising defects with free undermining and wound closure.

Nerve grafts are occasionally required following loss of substance of the facial nerve or nerves in the hand. Autografts are required and have been described by Bunnell, Ballance, and Duel.

support to maintain its shape. The curvature desired may be obtained by using an appropriate site on the helix. A defect in the helix may be repaired by attaching a skin flap from the postauricular

region which is severed at its base about two weeks later, folded and sutured to the posterior edge of the defect. A full thickness section of the lobule of the ear may be grafted in the same way to restore nasal defects; but if rigid support is required, a strip of cartilage must be added later.

Skin Defects and Pedicle Flaps.—Removal of a scar, area of granulation tissue or a tumor may be accomplished by elliptical excision and immediate closure of the defect by suture. If the wound is not large, this

procedures, which are least complicated as to rotation of tissue and in which the length of the scar is minimal, are usually the most satisfactory.

A skin flap may be defined as a given amount of skin and subcutaneous tissue which is attached to the body at some part of its periphery, through which it receives its blood supply. This flap may be rotated from its original site to fill an adjacent defect and is called an *open flap*. The blood supply to a flap may be increased by raising part and

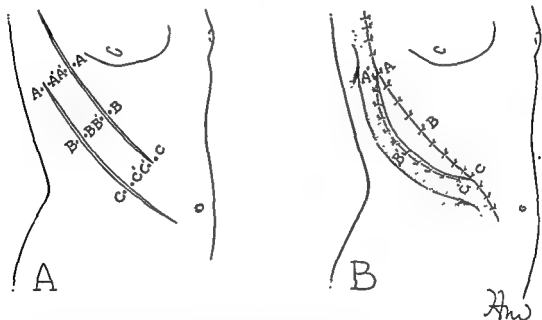


Fig 46—Method of formation of tubed pedicle flap.

A. Outline of staggered skin incisions to facilitate closure of the wound at each end of the tube. A number of points are marked opposite each other on the flaps to indicate the correct site for insertion of sutures to form the tube.

II Following free undermining, the wound left by formation of the pedicle is closed by sliding flaps of skin beneath the tube and suturing securely. If the defect is too wide, it should be covered with a split skin graft.

may be accomplished by undermining the adjacent skin and approximating the skin edges by suture. The resulting straight scar is most inconspicuous, particularly if it is parallel to Langer's lines of skin tension. Larger defects may be eliminated by converting the defect into a square, triangular or other geometric shape; then by undermining and freely shifting local skin flaps, the deficiency may be overcome. Those

then suturing it back in its bed. This is called a *delayed flap*.

A *tubed flap* eliminates raw areas and chronic infection present in open flaps. The desired width and length of the flap having been determined, parallel incisions are made through skin and fat and the strip of skin is *undermined*. Points opposite each other on the flap, previously marked with dye, are sutured with the skin surface outward. The

lateral skin edges are undermined and closed with sutures. If the tubed flap is too wide to permit closure of the raw wound by undermining, the raw area is covered with a skin graft. These flaps which form solid tubes of skin and fat attached at each end to the body may be transferred rapidly from one part to any other part or extremity, by transferring one end to the wrist or arm as a carrier. When the blood supply from the arm is adequate, the opposite end of the tube is severed and the whole flap is swung to the recipient site and sutured in place.

Z-Plasty.—Contraction of scar tissue may cause limitation of motion of joints, in many cases a heavy ropelike cord of scar tissue forms in the line of tension of scar over the area affected. The heavy scar may be partially excised and by suitably placed Z-shaped incisions with transposition of the two flaps, the bowstring tension may be overcome. The area adjacent to the flaps should be extensively undermined. This procedure causes an increase in length of the contracted scar. In the case of long bridge scars, double

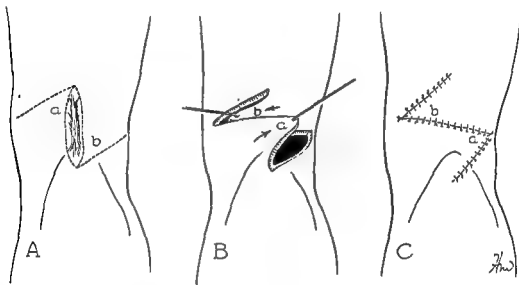


Fig 47 —Use of Z-plasty to relieve cicatricial contracture

- A The Z-incision with angles of about 60 degrees to obtain optimum increase in length
- B The flaps are transposed after free undermining
- C Skin closure with correction of the linear contracture

A *lined flap* is formed by folding together the raw surfaces of one end of the flap or by applying a skin graft to the raw undersurface of a flap. These are useful in repairing loss of two surface structures such as the cheek, lip, nose or eyelid.

An *island flap* consists of an area of skin and subcutaneous tissue attached and nourished only by blood vessels and a small amount of subcutaneous tissue. This flap is often used to transplant an island of scalp to reconstruct an eyebrow.

or triple "Z" incisions may be made in series with still greater relaxation resulting than that obtained by a single incision. When broad scar bands have formed, for example, in chest and arm adhesions following severe burns, the shifting of local tissue flaps may be combined with free skin grafts.

A **Hypertrophic Scar** is one in which there is excessive formation of fibrous tissue and is often seen following deep second and third degree burns or infected wounds. Many of these scars subside spontaneously in time or

their departure may be hastened by a course of x-radiation.

A Keloid may be easily recognized since the growth of fibrous tissue extends beyond the original limits of the wound and sometimes occurs following minimal trauma. Excision and skin grafting followed by early irradiation appear to be the most effective treatment.

CONGENITAL AND ACQUIRED DEFECTS

Some children have the misfortune to be born with congenital defects of various types. Cleft lip and palate will be discussed in a later section. A rather common congenital defect in children is lop ears, quickly noted by playmates who ridicule the victim

A.



B.



C.



D.



Fig 48—A and B Protruding ears which are large, show no antihelix and stand out at right angles to the head

C. and D. Postoperative result achieved by the operative procedure described. Note the reconstruction of the missing antihelix

until an inferiority complex has been developed. Fortunately, this condition can be corrected by a simple operation which may be performed under local anesthesia. An

ellipse of skin is excised with its long axis at the cephaloauricular junction and a small ellipse of cartilage is also removed where the new antihelix is to be formed. The cut



A



B

Fig 49—A Marked saddle nose following childhood infection of septum
B Reconstruction with cartilage graft.



A



B

Fig 50—A Long hump nose with a drooping tip

B Six months after operation. The hump was removed, septum was shortened, and the alar cartilages were reduced in size.

edges of the cartilage are infolded with sutures and following closure of the skin incision a pressure dressing is applied. Excellent cosmetic results may be obtained as well as relief from the inferiority complex.

Carved autogenous cartilage transplants or cancellous bone from the ilium are most satisfactory materials for reconstruction of a saddle nose. The graft is carved to the proper size and is inserted through an incision in the tip of the nose. Grafts of bone should be brought in contact with bone by sliding the upper end under the periosteum,

create an unesthetic appearance. Obstruction to respiration should be corrected before or if feasible at the time of plastic reconstruction of the nose. Local anesthesia is the anesthetic of choice for rhinoplasty. Following the usual skin preparation and draping, incisions are made between the alar and upper lateral cartilage on each side and extended medially to separate the columella from the tip of the septum. The skin is now separated over the rest of the nose. The hump is removed with a saw at a predetermined level, depending on the individual case

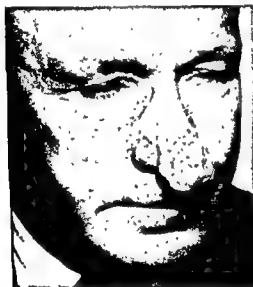


Fig. 51.



Fig. 52.

Fig. 51—Rhinophyma increasing in size steadily and causing patient to stay at home during the day to avoid embarrassment.

Fig. 52—Postoperative result after raising skin flaps, excising hypertrophied sebaceous glands and resuturing flaps after trimming away excess skin.

otherwise they tend to be absorbed. If tip support is required, a separate strut of bone or cartilage is inserted in the columella and attached to the piece of graft restoring the bridge. After closing the incision, a nasal splint is then applied to control swelling. One disadvantage of the use of bone grafts in the nose is the subsequent lack of normal resiliency of the nasal tip and the possibility of fracture of the graft.

Congenital or acquired deformities of the nose may interfere with function, as well as

and the upper lateral cartilages are trimmed to the same level. The nasal bones are separated with a saw from the septum. At this stage the septum should be shortened and the alar cartilages reduced in size if necessary. The nasal bones are now cut on each side at their junction with the maxillary bones and fractured. The columella is sutured to the tip of the septum with dermal sutures, and a nasal splint is applied. This is changed when necessary and discarded in a week or two.

Rhinophyma begins with acne rosacea and terminates with tremendous hypertrophy of the sebaceous glands often completely obliterating the normal contour of the nose. Several methods of treatment have been suggested.

1 Flaps of skin on the nose may be raised, the hypertrophied glands excised completely, and the flaps resutured after trimming away any excess skin.

method possesses certain advantages, and all factors should be considered in selection of treatment for each case.

Mammoplasty.—Individuals with pendulous breasts either flabby and atrophic or markedly hypertrophic sometimes request a plastic operation. The breasts should be free from evidence of malignancy, no serious systemic disease should be present, and there should be no history or evidence of psycho-

A

B



C

D

Fig 53—A and B Marked fatty hypertrophy of breast causing backache because of weight and difficulty in obtaining attractively fitting clothes

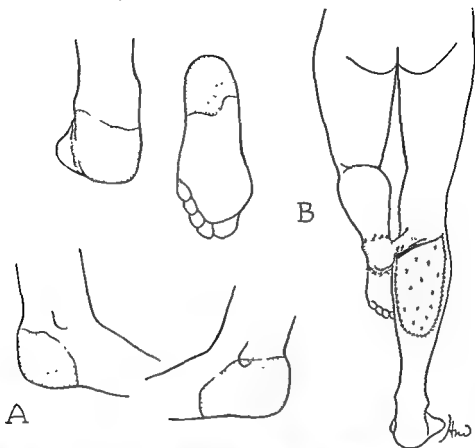
C and D Result one year after operation with relief of symptoms. Note cone-shaped breast contour

2 Complete excision of affected tissue and application of a free skin graft

3 Finally the growth may be pared down to the normal contour of the nose without removing all regenerative elements

Re-epithelization from remaining sebaceous glands occurs in about ten days. Each

sis. Many operations have been devised and some are suited to particular types of cases. Massive hypertrophy of the breast is easily treated by reflecting skin flaps after making an inverted "T" incision, amputating all excess breast tissue and fashioning the remaining substance into a cone shape by in-



C

D

Fig 54.—A. Illustration showing avulsion of soft tissues of heel when foot was run over by train

B Cross-leg pedicle flap after being delayed was swung over into defect on foot

C and D The result one year postoperatively showing flap. It is still protected by sponge rubber pad in shoe

interrupted sutures. The skin flaps are approximated to form a tight skin brassière which supports the breast. Excess skin is excised and the wounds are closed with fine sutures. The nipples which have been excised are replaced at esthetically suitable levels, which were marked before operation. A circular patch of epithelium is excised with a sharp scalpel, and each nipple is sutured carefully to the dermis. A firm supportive dressing is applied. A second type of operation commonly used in smaller breasts is removal of the required amount of the lateral part of the breast by an S-shaped incision. The lower free end is rotated upward and medially and sutured to the upper end of the incision in the gland, thus re-establishing the cone shape of the breast. Each nipple is transposed to a predetermined new level and brought through a circular hole in the skin which is the same size as the nipple. After replacing the nipple, draping, and suturing a tight brassière formed from the skin flaps, a pressure dressing is applied. In each case it is advisable to insert a small drain in the wound which is removed in 48 hours.

Meloplasty.—When weight is lost or with increasing age the elasticity of the skin is decreased and the skin falls into wrinkles. This sign of ageing is a matter of considerable importance for many, for business as well as social considerations.

These wrinkles may be removed from the forehead by incising in the hairline and undermining the skin down to the eyebrows. The excess skin is removed and the wound sutured. Baggy skin in the eyelids may be removed by incisions placed in wrinkle lines in the upper lid and one millimeter below the eyelashes in the lower lid. The incision for the face and neck starts in the hairline above the ear, curves in behind the tragus, emerges and crosses the lobule and up behind the ear to the hairline. The skin should be undermined close to the eye, nostril, and mouth and down into the neck. The skin envelope is drawn up tightly and all excess

is excised. Subdermal sutures as well as skin sutures are used to close the wound. A pressure dressing is applied for a week, and the face should be supported at night for a month.

Reconstruction of Extremities.—Loss of the thumb reduces the efficiency of the hand by one half. The thumb may be reconstructed by phalangization, pollicization of the index finger and use of local skin flaps or tubed pedicles with insertion of a bone peg for support. When skin and subcutaneous tissues have been avulsed from the hand exposing tendon and bone, it is necessary to cover the defect by shifting local flaps or applying a pedicle flap from another area. One of the most suitable sites is a flap from the lower abdomen, with the base in the inguinal region and which receives its blood supply from the inferior epigastric vessels. In chronic osteomyelitis of the leg, after all infected bone has been removed a split skin graft may be applied immediately to the cavity with a high percentage of take. When all sequestra have separated and infection is no longer present, the depressed area may be resurfaced with a cross-leg or other type of pedicle flap. The skin of the plantar surface of the foot may be avulsed in industrial or railway accidents, leaving bone exposed and infected. Free skin grafts will not suffice here since there is insufficient padding over the bone to permit weight bearing, and a pedicle flap with a good layer of fat usually from the opposite leg is necessary. Even after the flap has healed, the flap must be protected by the use of a shaped, sponge rubber pad worn in the shoe because the flap will never acquire the special histologic structure of normal plantar skin.

CLEFT LIP AND PALATE

Introduction.—Egyptian mummies have been found showing this congenital deformity, and Celsus is credited with being the first to repair cleft lips by paring the margins and suturing the wound. According to the

latest surveys it has been found that cleft lip or palate occurs once in from 750 to 850 living births in a Caucasian population, although the incidence is only half as frequent among Negroes. A cleft lip is approximately twice as often situated on the left side and is more frequent in males.

Embryology.—The classical concept of development of the component structures of the lip, alveolar process and palate by growth and subsequent coalescence of five various processes about the primitive oral cavity has been questioned by a number of investigators during the past thirty years. According to Veau there are no processes and clefts. The nasal orifices are formed by invagination of the surface ectoderm. The ectoderm is invaded by the mesenchyme growing into the lip and subnasal region from the lateral part of the face. Thus the primitive primary palate and lip are formed. Only in the posterior palate is there fusion of processes from either side. Failure of mesenchymal penetration at the 11 mm stage may thus result in various types of cleft lip or palate, due to rupture of the thin epithelial partition by the strain of constantly growing facial structures. Recent work of the school of experimental embryologists shows that specific organs and tissues are induced by "organizers" and also by the action of various chemical substances upon embryonic cells. These organizers act on embryonic cells and alter their pattern of growth. The development of the optic cup from the neural plate for example, is induced by a portion of the archenteric roof immediately beneath it. It has been shown that any lack of direct contact of the cells with the organizer will prevent or modify the development of specific tissues or organs.

Etiology.—The precise cause of this condition has not yet been established with certainty. While the commonly suspected conditions of alcoholism, syphilis, falls during pregnancy, and malposition of the fetal hands or tongue have been blamed, there is no doubt that these can be excluded in favor

of other possibilities such as vitamin or other dietary deficiencies as well as rubella and acute infections occurring during the first trimester of pregnancy which may exert an adverse effect on the product of conception. It has been claimed that pregnancies occurring late in the reproductive life of the mother tend to produce a greater number of children born with congenital defects. A recent study does not confirm this conclusion. It must not be forgotten, moreover, that there is a definite positive family history in from 20 to 30 % of cases. Warkany believes that cleft lip and palate are inherited as a double recessive trait, one gene being autosomal and the other sexlinked. According to Fogh-Andersen, cleft lip with or without cleft palate should be considered hereditary, whereas other factors are responsible in isolated cleft palate. It has been found that offspring of pregnant mice treated with cortisone show a high incidence of cleft palate as well as other defects.

Types.—The cleft of the lip may be unilateral or bilateral, and these may be partial or complete. A cleft palate may involve merely the uvula or extend into the soft palate, hard palate, and finally through the alveolar ridge, when it is associated with a complete cleft lip and palate. In the simplest variety the cleft of the lip may result in notching of the vermillion border, and there is usually a muscular defect and asymmetry of the nostril on the affected side. When the condition is more marked, the notching of the lip extends for a varying distance toward the nostril and there is frequently notching of the alveolar process. In yet another type the cleft involves the floor of the nostrils; the nostril is markedly widened and the alveolar process is cleft.

Operation.—The opportune time for operation is between 6 and 8 weeks after birth. There are those who advocate operation within the first week of life, but since there is never an emergency due to feeding problems, there is no reason for not taking advantage of the better condition of the patient

A



B



C

Fig 55—A *Thompson operation (modified)* A and A' are marked with methylene blue at the base of the columella and the nostril but retaining sufficient tissue on each side to form a floor for the nostril equivalent in width to the opposite nostril. The vertical length of the lip on the normal side is measured with a compass from the base of the nostril to the vermillion border of the lip. Fixing the regulating screw at this length, measurements are taken in the lip at each side of the cleft commencing at A and A' and extending to B and B'. The latter points are placed at the junction of the skin and vermillion of the lip. The margins of the cleft are now denuded with a single stroke of a narrow-bladed scalpel. The nostril and skin of the cheek on the cleft side are undermined freely as is the internal lining of the skin of the ala and tip of the nose. Correct relationship of the external and internal skin surfaces of the ala is maintained by through-and-through mattress sutures tied over a small piece of Xeroform gauze. The muscle layer of the lip is approximated with several 00000 catgut sutures. The skin edges are sutured with interrupted No. 40 stainless steel sutures and after excising the excess vermillion in the form of double V-shaped flaps, these are imbricated and sutured with 00000 Dermalon sutures. A Logan lip bow is then applied to immobilize the lip.

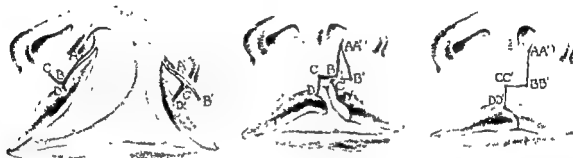
■ Appearance of preoperative partial cleft lip without palatal defect.

C. This procedure simulates the normal philtrum and in suitable cases provides a full well-shaped lip.

and larger and more easily handled tissues of the lip when the child is somewhat older. Consistently better results can be obtained when the child is in good condition and has reached a weight of 9 or 10 pounds. The ideal operation should result in a nostril of the size, shape and position similar to the

opposite side; a full, loose lip and a symmetrical vermilion border, simulating a cupid's bow if possible, and a faint scar. Prior to operation the child should be free of obvious respiratory infection and the skin about the lip should be clean. The most satisfactory anesthetic is endotracheal gas and

A.



B

C.

Fig. 56—A *Hagedorn-LeMesurier Operation*. The points A and A' are located as in the previous method. The point D' is selected on the mucocutaneous border of the cleft side as high up as possible where the mucous membrane is adequate in thickness. From this point a line D'C' is extended perpendicularly from the vermilion border and varies from 3.5 to 4.5 mm in length depending on the age of the child. The lines A'C', C'B' are of equal length. The latter curves slightly toward the vermilion border. This produces a moderate pouting of the lip which is a normal characteristic. On the medial side a line AB the same length as A'B', is drawn down to the vermilion border and a line CD is extended into the lip at right angles to the mucocutaneous border. This is the same length as C'D'. Incisions are then carried out as shown in A and after free undermining of the cheek and lining of the ala the flaps are approximated as illustrated in A. The muscle layer and skin edges are sutured with fine catgut and wire as previously described.

B and C.—Pre- and postoperative photographs showing the full pouting lip which is obtained

oxygen supplemented, when necessary, by ether. This provides the operator with sufficient time for a leisurely and planned operation. If the hemoglobin is lower than average, a blood transfusion should be given of from 100 to 150 c.c., and this may be administered through the femoral vein or into the long saphenous at the level of the internal

malleolus. Since the type of deformity varies considerably, it is helpful to employ the Thompson, Mirault-Blair or Hagedorn-Le-Mesurier procedures when indicated, depending upon the type of cleft.

Bilateral Cleft Lip.—This type of deformity requires quite a different procedure from those employed in the single cleft lips, in



Fig 57—A *Operation for Bilateral Cleft Lip*. The incisions ABC are outlined on both sides of the cleft and on the philtrum. After very extensive freeing of the nostril and cheek from the maxilla on both sides, suture of the muscle layers and skin margins is carried out as described in single cleft. The vermillion border is imbricated and a Logan lip bow is applied. A full well-balanced lip is obtained by repairing both sides at the same time.

B and C. Preoperative and postoperative photographs

order to avoid unsightly tight lips, too frequently seen. The premaxilla, if protruding, should never be replaced posteriorly by excising a wedge of septum or even by splitting it obliquely and sliding the premaxilla back. In the past, this practice has been one of the most common causes of severely retracted upper lips. The object to be achieved is the displacement of the premaxilla posteriorly by traction of the repaired lip muscles, causing it to assume a relatively normal relationship to the maxilla. Marked interference with its blood supply will inhibit normal growth. In this type the cleft may be complete or incomplete, as in the single lip. Following operation for cleft lip the suture line is kept clean by swabbing with cotton-tipped applicators dipped in penicillin solution 1:1,000 until bleeding and crust formation ceases.

The baby may be fed by a small glass syringe or may be allowed to drink from a small cup or glass. The sutures are removed as indicated, starting on the fourth day. Application of arm splints for the entire postoperative period will prevent the child from injuring the healing lip.

Cleft Palate Surgery

The goal of cleft palate surgery has long since advanced from the concept of merely providing an intact partition between the oral and nasal cavities. With improvement in technique it has become obvious that the function of normal speech is the aim to be achieved in every case. This must include the services of the speech therapist, orthodontist, and, if necessary, the prosthodontist. The ability of the soft palate to move freely upward and backward and close the nasopharyngeal sphincter is the test of a successful operation, and those procedures which may interfere with this function of the soft palate should not be employed. In 1764, Le Monnier, a French dentist, reported the first successful repair of a cleft velum.

The optimum time for closure of the cleft palate is between 18 and 24 months. A cleft

palate does not materially affect the nutrition of the child, although a higher incidence of middle-ear or sinus infections occurs in these individuals. This probably results from the admission of oral contents into contact with the nasal passages. The types of operations most commonly carried out are as follows:

The von Langenbeck Procedure.—Longitudinal relaxation incisions are made near the teeth. The mucoperiosteal flaps are raised from the hard palate and elevated toward the midline after severing the palatal aponeurosis and fracturing the hamulus. These may be felt as small bony prominences in the anterior and lateral part of the soft palate on each side. The margins of the cleft are split or carefully pared, removing only the mucous membrane. The nasal side of the mucous membrane is then approximated with a number of interrupted catgut or dermal sutures with the knots projecting into the nasal cavity. A large horizontal mattress suture is carried from the lateral border of the soft palate, on one side, to the opposite region where it serves as a restraining hammock to splint the muscles of the soft palate during postoperative healing. The margins of the cleft in the oral cavity are then approximated with horizontal mattress, dermal sutures from the anterior termination of the cleft and extending into the soft palate and over the posterior aspect of the uvula.

The patient is fed on a liquid diet, gradually changing over to a soft diet at the end of a week. The sutures are removed in approximately two weeks and subsequently normal diet may be resumed.

Other Procedures.—In clefts involving only the soft or part of the hard palate, a U-shaped incision may be made medial to the teeth from one maxillary tuberosity to the other and the whole mucoperiosteal flap which has been raised may be set back after stretching the major palatine arteries from the posterior palatine

foramen The cleft which is present is closed at the same time in the manner previously described

posed on the nasal surface of the palatal flap may be skin grafted in an effort to minimize scar contracture of the raw surface.

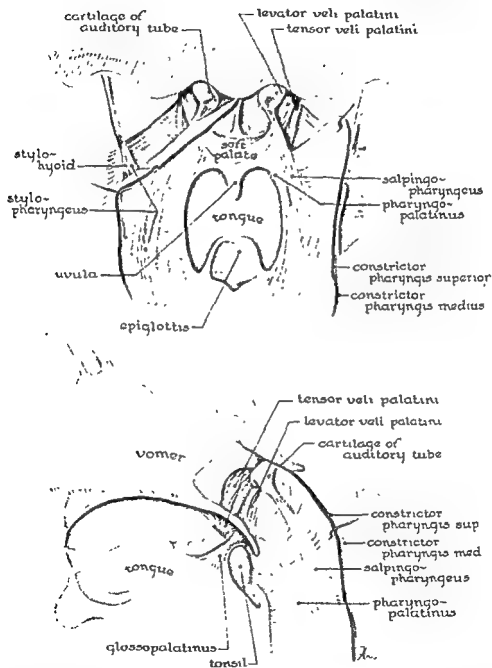


Fig 58.—Posterior and lateral views of the palatal muscles are illustrated

Occasionally, a "set back" may be performed secondarily on an intact repaired palate which is too short to permit good speech function. The raw surface thus ex-

In complete clefts of the palate, another procedure which is of great value is the vomer flap by means of which the cleft in the hard palate is closed by inserting a flap

A.



B.



C.

Fig 59.—A. *Dieffenbach-von Langenbeck Operation*. Through lateral incisions, mucoperiosteal flaps are raised from the hard palate with a curved elevator. The major palatine artery is spared. The palatal aponeurosis is severed and the medial margins of the soft palate are split or pared so that an even raw surface is prepared for suturing. The hamulus may be fractured but only if necessary to secure adequate mobilization of the soft palate. The nasal mucosa is sutured in a separate layer with fine catgut sutures so the knots project into the nasal cavity. The muscle layer and oral mucosa are then sutured with horizontal mattress sutures of Dermal including both surfaces of the uvula. A single large stay suture may be inserted in the soft palate if desired.

B. Cleft of palate to alveolar ridge is shown

C. Healing of palate with minimal scarring following von Langenbeck operation. No speech therapy was required

of mucosa from the vomer underneath the edge of a mucoperiosteal flap which has been raised on the opposite side of the palate.

Persistent small perforations which occasionally occur following a palatal operation, due to separation of the suture line, may be closed by freshening the margins of the perforation and resuturing the marginal defect after relaxation incisions have been made.

Following operation on children with cleft palate many require special care to achieve the utmost measure of rehabilitation. Frequent dental examination and filling of cavities in both deciduous and permanent teeth are necessary to avoid extensive caries and formation of dental abscesses.

Orthodontic treatment begun early may be necessary properly to align irregularly situated teeth or expand narrow dental arches. When palatal tissues have been extensively scarred, prostheses with an extension to partially occlude and aid normal closure of the nasopharynx may be necessary. Secondary adjustment of scars or contour defects of the lip, or nose should be performed when the child is old enough so that the maximum permanent improvement in appearance will be obtained. Speech therapy is frequently necessary to obtain the optimum improvement in speech and should be started as soon as the child is four or five years old.

TRAUMATIC INJURIES OF THE HEAD AND FACE

Intracranial and Facial Injuries.—In severe motor accidents and other types of major trauma to the head, it is always important to consider the possibility of intracranial complications such as cerebral or extradural hemorrhage, cerebral concussion with edema, or contusion and laceration of any part of the brain. Among the signs which should be noted are periods of unconsciousness, either immediate or developing after a lucid period; vomiting; changes in pulse, temperature and blood pressure; increasing papilledema; and loss or hyperac-

tivity of various reflexes throughout the body. The patient may be restless or become violently disturbed and require restraints. Further signs of severe cerebral injury are indicated by a leakage of cerebrospinal fluid from the nose or of blood and spinal fluid from the ears. If at all possible, the collaboration of a neurosurgeon should be obtained so that intracranial procedures may be carried out if necessary without delay.

With the exception of a terminal condition, there is no contraindication to the taking of roentgenograms provided reasonable precautions are taken. Morphine, however, should not be given, since it would mask the symptoms essential in diagnosing the patient's condition. At this stage it is usually wise to practice conservative care and to reduce and splint only those fractures which may cause obstruction to respiration or prevent control of hemorrhage. Supportive treatment, careful nursing, and maintenance of a free airway are the triad of care on which a successful outcome of many of these cases depends. After the first 24 hours, signs of meningitis may develop as a result of tears in the dura—usually in the region of the ethmoid bone or frontal sinuses—although basal fractures not infrequently may result in infection in the middle fossa extending through the external auditory meatus. It is important to perform a careful examination of the cranial nerves since medicolegal difficulties may arise should the patient claim injury to any of the cranial nerves following surgical intervention.

Scalp Injuries.—Scalp wounds may vary from small lacerations to partial or complete avulsion. In the former group it is usually only necessary to shave the scalp about the wound after carefully cleansing the margins and deep recesses of the wound. The wound may be closed with interrupted sutures of wire or nylon. The application of a firm pressure dressing will prevent the formation of a hematoma, and usually makes stab drainage wounds unnecessary. Deep burns

of the scalp may cause bald areas which may be eliminated by repeated partial excision and closure by undermining or transposition of flaps. Not infrequently, large or small areas of the scalp may be avulsed. These should be treated as soon as possible. When the area is no larger than two to three inches in diameter, it is possible by undermining or shaping of local flaps, to close the defect immediately without undue tension. Closure is facilitated by making relaxing in-

Lacerations and Other Injuries.—Facial lacerations are very common and their etiologies diverse. Many are due to accidents in the home. Some are the result of industrial accidents while others occur following crashes of transport vehicles and sporting accidents. Because of their frequency, certain basic principles should be observed if facial disfigurement or functional deformity resulting from inadequate treatment is to be avoided.



Fig 60—A. Patient was struck on head with roller from printing machine which caused concussion, fracture of temporal bone, frontal bone communicating with the frontal sinus, complete separation of malar bone from all bony and soft tissue attachments and contused wound of soft tissues

B The frontal bone was replaced, the malar wired to the zygomatic arch and frontal bone, and the wound closed with fine sutures after careful cleansing and minimal excision of soft tissue. Result six months postoperatively (This case was treated in conjunction with Dr. Arthur Elvidge.)

cisions in the galea to permit a more complete stretching of the scalp. On the other hand, in the case of extensive or total avulsion, the pericranium should be cleansed and dermatome skin grafts (0.016 to 0.18 inch thick) should be applied without delay. It has been found that by the local and general use of antibiotics and scraping of purulent exudate from the surface, it is possible successfully to skin graft wounds of this type up to 48 hours of injury.

Primary measures following serious facial injuries are control of hemorrhage, maintenance of adequate airway or provision by tracheotomy and treatment of shock by transfusion of blood or plasma. The blood supply of the soft tissue of the face is excellent, and there appears to be increased resistance to infection which permits healing even with unfavorable circumstances. Local anesthetic is most satisfactory in uncomplicated cases. With the advent of new

antibiotics, capable of inhibiting a wide spectrum of bacteria, clean-cut lacerations may be closed by primary suture 24 hours after the accident. Tetanus antitoxin or a booster dose of toxoid, if the patient has been actively immunized, should be administered.

Preliminary examination should determine the condition of the parotid ducts, the branches of the facial nerve, and the possible disturbances of vision, such as diplopia, or interference with ocular movement. Subsequently, the frontal, nasal, zygomatic, maxillary and mandibular bones should be palpated gently to form an estimate of the extent of the injury.

Permanent tattoo marks will result if these foreign substances are not removed completely.

Lacerations of the face should be carefully cleansed with soap and water and hydrogen peroxide or aqueous Zephiran. The recesses of the wound should be minutely explored for embedded glass or foreign bodies. In these cases only filamentous epithelial margins or small, jagged edges should be excised since scars are invariably finer when no débridement as is commonly understood, is carried out. It is useful, in many instances, to excise a small wedge of the dermis on each side of the wound so that



A.



B

Fig 61 —A Large trap-door flap of cheek with base situated inferiorly. The large metal missile which created the wound also caused a depressed fracture of the malar bone.

B The depressed malar fracture was elevated directly through the wound, following which careful cleansing and suturing of the wound were performed. Result one year post-operatively.

Wounds of the face fall into various categories. One of the common results of automobile accidents is the presence of road burns with deeply imbedded dirt and oil ground into the tissues which may be removed only by careful scrubbing with a small brush with soap and water or some detergent. Only when the wound appears clean to close inspection should it be dressed with Xeroform gauze and a pressure dress-

when continuous, subcuticular sutures of No. 40 gauge, stainless steel wire or Dermalon are inserted in the deepest part of the dermis, the margins of the wound will pout slightly. The epithelial margins of the wound are then carefully approximated with interrupted sutures of wire or Dermalon and tied without tension, this will enhance eversion of the wound edges. These sutures should be removed by the third or fourth

day since the wound is supported by the layer of deep sutures

In a kinetic region, it has been shown that a stretching and widening of the scar which often develops gradually over a period of a few months is minimized, since the everted wound edges are first drawn level before the margins can separate to form an unsightly scar.

Lacerations caused by glass are prone to create large trap-door flaps with long bevelled thin margins which form a lumpy, elevated flap on healing due to contracture. This effect may be minimized if as much of the bevelled edges as possible is removed in an effort to convert the injury to that of a simple incised wound. Furthermore, the lengthy use of a firm pressure dressing over the flap for three or four weeks during the period of fibrotic contraction tends to minimize its tendency to contracture.

Through-and-through wounds involving the ear, lip, cheek, nostril or eyelid should be approximated, in layers, with the utmost accuracy, and should remain immobilized in a pressure dressing for sometime to permit complete healing. The intermediate structures should be approximated with sutures, as well as the mucosa and skin surfaces. For example, cartilage of the nose or ear should be replaced into its normal position and retained with fine interrupted No. 40 gauge sutures. Severed muscle of the lip or cheek should be retained in a similar manner to avoid the creation of a muscular hiatus which will cause a most unpleasant cosmetic defect.

A small defect of the face caused by avulsion of tissue may be repaired by undermining the adjacent tissue with direct suture or by sliding or rotation flaps. A large defect with a vascular bed may be dressed either temporarily or permanently with a free skin graft. If a large portion of the cheek, lip or nose, has been avulsed, the skin and mucosal surfaces should be sutured to prevent distortion and contracture while definitive treatment is planned.

Occasionally, a portion of the ear, nose or other feature may be severed completely. If the part can be retrieved and resutured in position within a period of a few hours after injury, there is a fair possibility of the fragment taking as a free graft. Use of a pressure dressing and systemic antibiotic therapy are important adjuncts. If seen early, wounds of the floor of the mouth and tongue may be sutured primarily. Systemic therapy with antibiotics should be used. If Stenson's duct has been severed and diagnosis is made at the time of the injury a filiform catheter may be inserted through the duct from the mouth into the severed posterior portion and the cut ends approximated over the catheter with fine sutures. The filiform may be removed in a few days. Chronic fistulas resulting from injury to the parotid gland or duct are most simply and effectively treated by application of a pressure dressing for several weeks and avoidance of a diet and substances during the period of healing which cause marked salivary secretion.

Deep lacerations may cause facial paralysis from severance of the main trunk or branches of the facial nerve. At time of injury an attempt should be made to locate and suture the nerve ends if possible because secondary procedures are more difficult.

Fractures of Facial Bones.—These fractures should be immobilized as soon as the condition of the patient permits, since more accurate reduction of the fragments is possible, the patient is more comfortable, and healing without complication is more likely to occur. Laceration of the soft tissues if combined with fractures of the facial bones may greatly simplify reduction of the fracture, following which the soft tissues may be sutured. Comparatively simple operative procedures or methods of splinting suffice in a large proportion of facial fractures.

Nasal Bones.—Fracture of nasal bones usually causes bleeding from the nose, nasal obstruction, ecchymosis of eyelids and frequently visible deformity. One nasal bone may be depressed, there may be a fracture dislocation of both bones and septum or the

nasal bones and frontal processes of the maxilla may be comminuted and flattened. Simple fractures of the nasal bone may merely require elevation of the bone by means of a rubber sheathed hemostat within the nose, assisted by external molding with the fingers. When the bone is extensively comminuted and depressed backward between the frontal processes of the maxilla, in addition to elevating the bones and straightening the crumpled septum it will usually be necessary to support the bridge by means of

the upper lip, limitation of movement of the mandible, and diplopia. Fractures of the malar bone may result in a depressed fracture of the zygomatic arch or in the body of the malar or the bone may be extensively comminuted. In the first two varieties the bone may be elevated through a small incision made in the temporal region. A heavy, curved elevator is passed downward deep to an incision in the temporal fascia, and by an upward prying motion the bone may be raised to correct position. When the bone is



Fig 62—A The bridge of the nose is crushed and displaced down and backward, forcing the medial canthi apart. Deep lacerations expose the bony fragments and nasal passages.

B After elevation of the bony fragments and replacement of the fractured septum they are retained by a double wire sling passing through the nose and tied over aluminum plates on each side.

C Postoperative appearance

two No. 28 gauge wire sutures passed through the nose beneath the fragments from one side to the other. The wires are tied over lead plates so that the whole acts as a hammock as well as a splint, thus elevating the bridge of the nose and compressing the multiple fragments together.

Malar Bone.—An obvious symptom is a flattening of the malar region on the affected side although subsequent edema may conceal this feature. There may be anesthesia of

comminuted, it is necessary to expose the fragments through an incision in the mouth, and after passing a wire through a hole in the body of the bone the ends are brought through the cheek and tied to an outrigger projecting from a plaster headcap. This procedure may be supplemented with gauze packing in the maxillary sinuses for a week.

Maxilla.—The most characteristic feature of maxillary fractures is mobility of a segment of the upper teeth or the entire middle

third of the face, which is usually elongated. The teeth do not occlude and the patient cannot masticate food. There may be associated fractures of the malar and nasal bones with leakage of cerebrospinal fluid through the nose. The commonly observed types of fracture of the maxilla are fracture of one half of the maxilla; fracture of the anterior maxilla through the floor of the maxillary sinuses; and fracture of the anterior maxillary compound which usually involves one or

pyramidal type of fracture usually involves applying a rather complicated apparatus in which the maxilla is maintained in correct position by means of a Kingsley splint fastened to a plaster headcap by adjustable metal rods.

The period of fixation of fractures of the maxilla varies from three to eight weeks, depending upon the type of fracture and possible complications which may have developed.

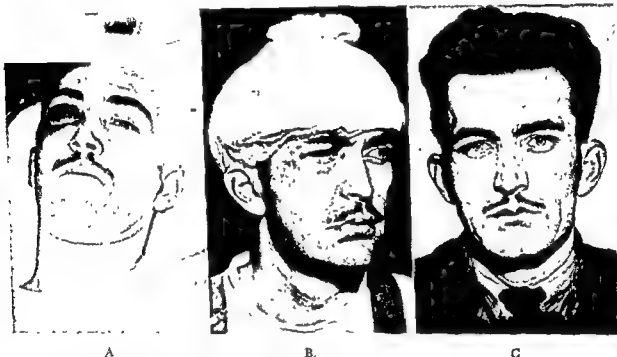


Fig. 63—A Depressed fracture of the right malar bone 10 days after accident.

B After freeing and elevation through the mouth the bone did not remain in position, so a No 26 gauge steel wire was passed through a hole drilled in the body of the bone, carried through the cheek, and attached to an outrigger bar on a plaster headcap which provided constant support for two weeks

C Result two months later

both of the malar bones and traverses the nose into the region of the glabella. The first and second varieties of fractures merely involve placing the upper and lower teeth in correct occlusion after the reduction of the fracture and wiring the jaws together. In these cases much comfort and necessary support will be afforded by a firm bandage which encircles the head and jaw. The

Mandible.—Fractures of the mandible are usually classified according to anatomical location and may occur at the symphysis, body, ramus, neck of condyle or coronoid process. Quite commonly fractures are bilateral and occasionally three or four separate fractures are present. Gunshot wounds may result in large segments of the mandible or of any of the facial bones being

A.



B.



Fig 64—A Pyramidal fracture of maxilla, nasal, ethmoid and frontal bones, with displacement posteriorly. Displacement of maxilla upward and backward is shown by the position of the teeth

B Result following reduction of maxilla and retention with intermaxillary wiring and reversed Kingsley splint

C X-ray showing extensive comminuted fractures of nasal, frontal and ethmoid bones. Note aerocoele from air which has entered through the fractured cribriform plates and torn dura

C.

blown away together with some of the soft tissues.

The appearance of a patient who has suffered a recent fracture with displacement of the mandible is alarming to those who may not have had much experience with this type of injury, but if a fair number of the patient's maxillary and mandibular teeth are present, there is probably no other bone

or arch bars, and intermaxillary wires. It should be noted, moreover, that about 90 % of fractures of the mandible fall into this category.

When the lower jaw is edentulous, wires may be carried circumferentially about the mandible with the ends projecting into the mouth so that they can be twisted over the patient's lower denture or a substitute. The simplest way to circumscribe the mandible with wire is to carry one end of the wire down the outside of the mandible close to the periosteum, and through a $\frac{1}{8}$ " incision in the skin with a large, straight abdominal needle, it is then reinserted through the same skin incision and carried up on the inside of the mandible close to the periosteum until it perforates the mucosa on the inner aspect of the alveolar ridge. Pin fixation appliances such as the Roger Anderson splint or individually molded acrylic splints may be employed when it is necessary to treat the fracture without immobilizing the mandible.

Diet.—When the jaws are wired together it is necessary to feed the patient a diet of semifluid consistency. It has been found that even with a complete complement of teeth the patient can manage with little weight loss if a diet of approximately 3,000 c.c. of fluid and semifluid substances containing 3,000 calories are taken each day. It is advisable to supplement this diet with Casec or skimmed milk powder and add to the mixture a high multivitamin concentration which may be dissolved in the liquid feedings. Careful oral hygiene measures should be maintained throughout.

Anesthetics.—The anesthetic of choice is local, supplemented when necessary in the more complicated fractures by a short period of Pentothal anesthesia. Thus the troublesome complication of vomiting is rarely encountered.

Complications.—A tooth in the line of fracture should be preserved even if loose, since it may prevent displacement of a posterior fragment until sufficient time has elapsed to permit adequate callus to form. At the first sign of development of invasive

A.



B.

Fig 65—A Model showing the method of applying an cyclet loop of No 25 gauge stainless steel wire about teeth.

B Arch bars wired to upper and lower teeth and held together by intermaxillary wires. Displaced fragments reduced.

which may be so simply and accurately replaced following trauma. The maxilla, if intact, forms an exquisitely accurate counter die in which the separated fragments of the mandible and its contained teeth may be interlocked and retained with eyelet loops

infection the tooth should be removed. Osteomyelitis is rarely seen if antibiotic therapy has been employed. Adequate drainage should be provided and sequestrum formation awaited before removal is attempted. Nonunion may occur because of malalignment or loss of bone. The bone ends are freshened and wired in proper alignment or a bone graft may be inserted to replace missing tissue. Trismus may occur as a result of prolonged immobilization of the mandible or from scar tissue formation or bony obstruction. It usually disappears spontaneously, but mechanical dilating appliances may be used if required. Surgical removal of scar tissue or displaced bone fragments may be necessary in some cases. Ankylosis due to intra-articular bony or dense fibrous union may follow a blow on the jaw or a fracture. The condyle may be removed or a segment of bone a centimeter or more in width is removed across the width of the ramus below the condyle if it is solidly united.

Dislocation.—The usual dislocation is forward and may be unilateral or bilateral. Upward, backward, and forward dislocations may occur but are associated with fracture of the condyle. Forward dislocation may occur by yawning, vomiting, undue use of a mouth gag or a blow on the chin with the mouth open. Chronic recurring dislocation may occur if the temporomandibular ligaments become relaxed. Diagnosis is made by noting that the mouth is held open and fixed, chewing is impossible, and the condyle may be felt in front of the mandibular fossa which is empty. In unilateral dislocation the chin is deviated to the opposite side. Treatment consists of lifting the chin with the fingers while the thumbs protected in gauze are placed on the lower molar teeth and pressure is exerted downward. A general anesthetic may be necessary, if muscle spasm is present. To prevent recurrence, opening of the jaw may be limited by applying a chin bandage for three weeks or intermaxillary wires which will only permit excursion of a centimeter.

REFERENCES

Plastic Surgery

- Baxter, H., and Enten, M.: Clinical Study of Fate of Hemorrhage in Man. *J. Surg.* 81: 1169-1174, 1938.
- Davis, J. S., and Kitlowski, M. A.: A Method of Tubed Flap Formation, *South M. J.* 29: 1169-1174, 1938.
- Davis, J. S.: The Use of Small Deep Grafts in Repair of Surface Defects, *Am. J. Surg.* 47: 280-298, 1940.
- Hempelmann, L. H.: Acute Radiation Injuries in Man, *Surg., Gynec. & Obst.* 93: 385-403, 1951.
- Langer, K.: Zur Anatomie und Physiologie der Haut I. Ueber die Spaltbarkeit der Cutis Sitzungsber. d. k. Akad. d. Wissensch. Wien, Abt. I. 44: 19-46, 1861. II. Die Spannung der Cutis. *Ibid.* 45: 133-156, 1862. III. Ueber die Elasticitat der Cutis, *Ibid.* 45: 156-178, 1862. IV. Das Quellungsvermogen der Cutis, *Ergebnisse, Ibid.* 45: 179-188, 1862.
- Padgett, E. C.: Calibrated Intermediate Skin Grafts, *Surg., Gynec. & Obst.* 69: 779-793, 1939.
- Peer, Lyndon: Cartilage Grafting, *S. Clin. North America* 24: 404-419, 1944.

Cleft Lip and Palate

- Baxter, H., and Fraser, F. C.: Production of Congenital Defects in Offspring of Female Mice Treated With Cortisone: Preliminary Report, *McGill M. J.* 19: 245-249, 1950.
- Davis, W. B.: Harelip and Cleft-Palate: Study of 425 Consecutive Cases, *Ann. Surg.* 87: 536-554, 1928.
- Dorrance, G. M., and Shirazy, E.: The Operative Story of Cleft Palate, Philadelphia, 1933, W. B. Saunders Company.
- Fogh-Andersen, P.: Inheritance of Harelip and Cleft Palate, Copenhagen, 1943, Ejnar Munksgaards Forlag.
- Veau, Victor, and Borel, S.: La Division palatine, anatomie, chirurgie, phonétique, Paris, 1931, Masson et Cie.
- Warkany, J.: Etiology of Congenital Malformations, *Advances Pediat.* 2: 1-63, 1947.

Traumatic Injuries of the Head and Face

- Baxter, Hamilton, and Elvidge, Arthur: Neurological and Plastic Repair of Cranial and Dural Defect, *Canad. M. A. J.* 56: 202, 1947.
- Erich, J. B., and Austin, L. T.: Traumatic Injuries of the Facial Bones, Philadelphia, 1944, W. B. Saunders Company.
- Ivy, R. H., and Curtis, L.: Fractures of the Jaws, Philadelphia, 1931, Lea & Febiger.
- Padgett, E. C., and Stephenson, K. L.: Plastic and Reconstruction Surgery, Springfield, Ill., 1948, Charles C. Thomas.
- Smith, Ferris: Plastic and Reconstructive Surgery, Philadelphia, 1950, W. B. Saunders Company.

CHAPTER X

NEUROSURGERY

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THE SCALP

Much can be learned from an examination of the scalp. In *traumatic cases* areas of ecchymosis and swelling give information which may be valuable from a diagnostic and from a medicolegal point of view. In a practical sense a severe contusion may indicate underlying fracture. Marked edema may indicate fracture and even rupture of the dura mater. Hemorrhage and edema of the soft extracranial tissues may form a ridge round the edge of an area of contusion which may simulate a depressed skull fracture. This line of swelling can be smoothed out with the examining finger. The crepitation of loose bone fragments can sometimes be palpated.

A contusion in the temporal region should suggest the possibility of an extradural hemorrhage from the middle meningeal artery. Contusion over the general region of the venous sinuses suggests the possibility of subdural hematoma. Discoloration and swelling of the eyelids and periorbital tissues associated with the development of subconjunctival hemorrhage suggests fracturing of the base of the skull. If fracturing is slight, or if it lies at some distance, the discoloration may take a day or more to develop. The subconjunctival hemorrhage generally appears first in the temporal segment. In severe cases it may involve the whole area about the iris. In direct orbital or neighboring injury, periorbital swelling and subconjunctival hemorrhage appear immediately.

Hematomas, excoriations and lacerations of the scalp hidden by hair may easily be missed, and serious infection results, if neg-

lected. Ecchymosis of the mastoid region usually accompanies a fracturing of the floor of the middle fossa with linear extension into the petrous bone. In this case, bluish discoloration of the ear drum generally develops at once in severe cases, or it may take one or two days to appear if the fracturing is slight or lies at some distance.

Acute infections of the scalp are now uncommon, due to early diagnosis and improved methods of treatment. Swelling about the eyelids may be associated with some neighboring inflammatory or vascular lesion either extra- or intracranial, such as frontoethmoidal sinusitis, osteomyelitis of the frontal bone, cellulitis of the orbit, epidural and subdural abscess. Periorbital swelling also results from venous congestion in thrombophlebitis and arteriovenous aneurysm.

In *nontraumatic cases* the general appearance and contour of the scalp is also informative. Sebaceous cysts frequently occur. They may be removed if the patient desires and must be treated when infected. Epidermoid and dermoid tumors call for removal. They may arise in the cranial diploe and there may be intracranial involvement. Cranium bifidum with meningocele or encephalocele will be discussed later under the heading of congenital anomalies. Angiomatous lesions and congenital formations of the dermis can be treated according to standard methods.

THE CRANIUM

General examination reveals the size, contour and general shape of the head, the presence of hydrocephalus, craniostenosis, microcephaly, of gross skull defects, irregu-

larities, the relative size of the posterior fossa, and in infants the size of the fontanelles and the width of the suture lines. The intracranial pressure can be judged to some extent on palpation over the anterior fontanelle. An unusual prominence may indicate the presence of an osteoma, or meningioma. Special curiosities of cranial configuration may indicate such conditions as acromegaly or Paget's disease. Auscultation sometimes reveals a bruit which is due to an arteriovenous fistula and occasionally to a large neoplasm.



Fig 66—Discoloration and swelling of eyelids, subconjunctival hemorrhage, excoriations, and contusion of the side of the head in a case of fracturing of the skull

Roentgenological examination of the skull reveals the general texture of the bone, congenital lesions, such as cranium bifidum, biparietal foramina, platybasia and basilar impression, certain changes which result from increase of intracranial pressure, the beaten silver appearance, enlarged foramina for passing vessels and decalcification of the



Fig 67—Epidermoid cyst of the temporal bone

dorsum sella, the size and the shape of the sella turcica, disturbances of the endocrine system as in hyperpituitarism; osteomyelitis, tuberculosis and syphilis; Paget's disease; eosinophilic granuloma and Schüller-Christian disease; the presence of benign and malignant tumors involving the skull.

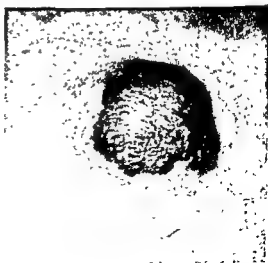


Fig 68—Angiomatous lesion of the scalp

A.



B.



C.



D.

Fig. 69—A. Microcephaly. B. Hydrocephalus. C. and D. Craniostenosis (note congenital biparietal foramina).

A



B



C

Fig 70—A Hydrocephalus in adult B Acromegaly, C Paget's disease

In trauma, in addition to the position and type of fracture and the state of the accessory air sinuses, one may derive help from

observing the position of the pineal gland and of the choroid plexus if these are visibly calcified. Occasionally one may find intracranial air, a traumatic encephalogram.

Osteomas may be removed when and if considered necessary by the surgeon, judged on position and rate of growth and desire of the patient. They grow very slowly. Their size can be checked periodically by x-ray. Dermoid cysts should be removed en bloc. Eosinophilic granuloma can be removed. Roentgen therapy alone has been used. The invaded bone in the case of a meningiomatous involvement must be resected at the time of removal of the whole tumor. For headache in Paget's disease one can decompress with a bi-subtemporal craniotomy. Encephalocele and meningoencephalocele can be repaired. Osteomyelitis can be cured by block removal including, if necessary, exenteration of the frontoethmoidal accessory air sinuses or mastoidectomy if either constitutes the primary focus.

THE MENINGES

The meninges are involved in the formation of congenital anomalies as in cranium bifidum and spina bifida with meningocele. The meninges may be involved in infection, acute, subacute, pyogenic, tuberculous, granulomatous, fungiform, and parasitic. Streptococcic, staphylococcic, and pneumococcic meningitis used to be frequently fatal. A few cases recovered from streptococcic meningitis by forcing fluids and adequate drainage by lumbar puncture or other means. Pneumococcic meningitis was 100 % fatal. Cases of staphylococcic meningitis rarely recovered. Since the employment of chemotherapy and antibiotics (sulfa drugs and penicillin) the mortality rate, even of pneumococcic meningitis, has been lowered to 30 %. In fact, in an otherwise uncomplicated and well-treated case, eradication of the primary focus and recovery should occur. Even often repeated daily lumbar punctures seem to be unnecessary except for exact evaluation of progress. At present streptomycin is being used in the treatment of tuberculous meningitis with some success. Occasionally one used to encounter pachymeningitis associated with a rapid spread of infection throughout the subdural space. This was invariably fatal.

Sometimes the subdural space may become primarily involved with the formation of a subdural abscess, and occasionally an extradural abscess may be encountered.

The case for subdural abscess has also improved. At one time from 60 to 100 % fatal, the results of treatment have improved from better appreciation of the condition, better surgical treatment, and the antibiotics. Infection in the subdural space, untreated, may spread over the whole central nervous system. It is always localized in the beginning and may remain so, arising from an infected accessory sinus or patch of osteomyelitic bone. Early diagnosis is difficult and important. Focal signs may appear, such as epilepsy, headache, and signs of in-

fection. The onset may be insidious. Treatment is by diagnostic trepanation. If pus is obtained, one anterior and one posterior skull trepanation can be made. The pus is evacuated by suction, and antibiotics are left inside the cavity. Drainage tubing or Penrose drains are suitably placed. These are left in for a variable period of days according to the type of case. With this local combined with general treatment the mortality rate is greatly reduced.

Extradural abscess must be treated by enlarged skull trepanation, evacuation, drainage, and use of antibiotics. A search for the primary focus of infection must be made and the source eradicated. Osteomyelitis or sinusitis must be dealt with appropriately.

The spaces adjacent to the meninges are of great importance in the pathology of trauma. The problem of subdural effusion and hematoma, and of epidural hematoma will be discussed under the appropriate heading. Tumors of the meninges will be discussed under the section on brain tumor.

CEREBROSPINAL FLUID CIRCULATION

The skull is a relatively rigid container and the brain within is lined and protected by three membranes. The *pia mater* which carries the blood supply for the cortex clings to its surface dipping into all the sulci. The avascular *arachnoid* bridges the small sulci but dips into the major clefts such as the median and horizontal sulci. Primitive mesenchyme separates with the coming of the cerebrospinal fluid into the *pia mater* and the *arachnoid*. The resulting space is known as the *subarachnoid space* and is continuous over the central nervous system. The *dura mater*, the outer tough sheath, is really composed of an inner more elastic and an outer fibrous layer. The *dura mater* clings more or less to the inner table of the skull. It splits along the midline of the skull and at the level of the tentorium and in the midline of the posterior fossa, to form the medial longitudinal sinus, the lateral sinuses

Left and right foramen of Monro

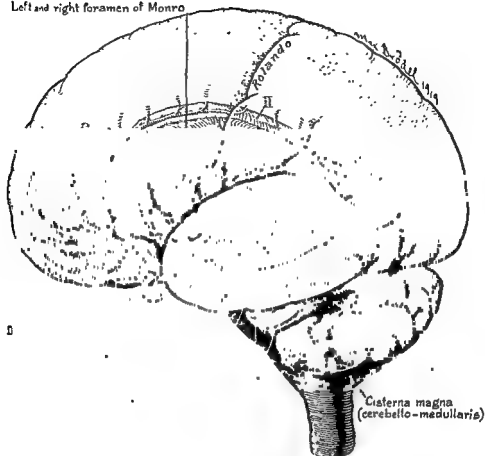


Fig 71 —The ventricular system (Dandy), and subarachnoid spaces through which the cerebrospinal fluid circulates. (From Dandy "Surgery of the Brain, Lewis' Practice of Surgery")

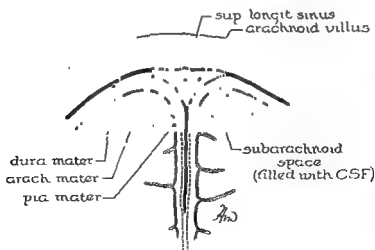


Fig 72 —Coronal section of the superior sagittal sinus showing relationship with adjacent meninges

and the occipital sinuses, respectively. It dips into the median cleft between the cerebral hemispheres as the *falk cerebri* and in the horizontal cleft between the cerebral and the cerebellar hemispheres to form the *falk cerebelli*.

Between the dura and arachnoid is the subdural space, which normally contains a little yellow fluid. In pathological states this potential space becomes of great significance as in the case of subdural hematoma and of subdural abscess.

Between the dura and the inner table of the skull there are generally some areas with little adherence. In certain injuries it is common for bleeding to occur from the middle meningeal artery and vein, which generally lie in and on the outer layer of dura. Extradural bleeding may strip the dura from the skull and give rise to an expanding extradural clot. Pus from an osteomyelitis may collect in the extradural space. Thus this area under pathological conditions assumes importance.

The cerebrospinal fluid is formed, few doubt, from the choroid plexuses and absorbed from the subarachnoid spaces through the vascular bed. Choroid plexus is present in the lateral ventricles. There is a smaller amount in the 3rd and in the 4th ventricle. Both clinical and experimental evidence shows that the fluid formed passes from the ventricular cavities by way of the foramen of Magendie and the foramina of Luschka into the subarachnoid cisternae and it is thought that much absorption takes place over the surface of the hemispheres.

Under healthy conditions arterial blood enters the cranial cavity at a certain normal pressure, e.g., 1,500 mm. of water and venous blood may leave with a pressure of 100 mm. of water.

The veins must carry a pressure above cerebrospinal pressure, otherwise they would remain collapsed.

The normal cerebrospinal fluid pressure is generally between 120 and 300 mm. water,

and true normal pressure might be considered to be 150 mm.

The normal contour of the ventricular and subarachnoid system may be visualized by the injection of air or oxygen into the spinal subarachnoid space or directly into the lateral ventricle. The indications for these procedures are beyond the scope of this summary; suffice it to say that generally speaking in cases of high intracranial pressure ventriculography is performed and in other cases encephalography.

If a block occurs in the ventricular system from any cause, be it inflammatory adhesion, congenital atresia of the aqueduct of Sylvius, or tuft of choroid plexus, tumor, parasitic cyst or abscess, there will arise dilatation of the ventricles proximal to the point of block. The result is internal hydrocephalus of a noncommunicating variety.

If blockage occurs in the subarachnoid space by adhesions, plastic exudate, diffuse neoplasia or hemorrhage, dilatation will occur not only of the ventricles but also of the subarachnoid cisternae posterior or proximal to the point of block. This is known as a communicating type of hydrocephalus.

If an expanding lesion arises in the brain it will cause distortion of the ventricular system and may cause some obliteration of subarachnoid markings, which may be seen in the ventriculogram.

If a contracting lesion or atrophic lesion is present, a passive increase in size of the ventricles and spaces may occur, and sometimes areas of focal dilatation of a ventricle give rise to an appearance of traction of a portion of the ventricle, called by Foerster and Penfield "ventricular wandering."

In cases of high intracranial pressure lumbar puncture can be a source of great danger. The pressure is released below but the intracranial pressure forces the brain into the foramen magnum causing herniation of the cerebellar tonsils which may result in strangulation of the blood supply to the medullary centers and death. In the case of a lesion above the tentorium a second

mechanism comes into operation. The uncinate gyrus of the temporal lobe may be crowded into the incisura tentorii adjacent to the cerebral pedicle, compressing the 3rd cranial nerve and even the posterior cerebral artery. The opposite or both cerebral peduncles may be notched against the sharp edge of the tentorium. Serious signs will arise from this type of herniation which include deepening coma, third nerve paralysis, decerebrate rigidity, and hemiplegia, which in 10 per cent of cases may be ipsilateral and which may be to some extent bilateral.

Compression of the third cranial nerve causes dilatation and fixation of the ipsilateral pupil, which is sometimes bilateral in terminal stages. Infarction of the occipital lobe from compression of the posterior cerebral artery and infarction of the brain stem may occur as late phenomena preceding death. These mechanisms operate in cases of high intracranial pressure. They are, on occasion, brought to acute fulfillment by lumbar puncture.

SYMPTOMATOLOGY

Symptoms and signs may be briefly discussed under two headings.

1. General symptoms and signs of increased intracranial pressure
2. Focal signs.

General Signs of Increased Intracranial Pressure

Headache may be generalized, bioccipital, bifrontal, severe and tending to become continuous. It varies to some extent with posture. Some believe that the headache is more severe in the morning. In final stages it is violent. Even raising the head from the pillow may accentuate it. In the rare case of a ball valve block at the isthmus of a ventricle, change of head posture may relieve or accentuate the headache. The history must be carefully taken to differentiate headache from other causes, e.g., migraine,

arterial hypertension, trauma, psychosis, low intracranial pressure, hypoglycemia, sinus disease, or eye strain.

Vomiting is due to a disturbance of centers in the medulla probably on a vascular basis due to pressure anemia.

Papilledema is present in the majority of cases of expanding lesions which have raised the intracranial pressure, although in many cases it is absent. Blurring of the edges of the disc first occurs on the nasal side and may rapidly invade the whole edge. Swelling generally develops in the same order and eventually the normal cup becomes a swollen convex surface. Hemorrhages and exudate often appear relatively late or not at all in these cases of increase of intracranial pressure. In arterial hypertension one generally sees more widespread patches of exudate and hemorrhage for the degree of papilledema present.

Clinically during this time the blind spot enlarges and the visual fields become constricted and visual acuity eventually diminished. If the pressure is removed, recovery, good or relative, will occur. If allowed to continue, blindness will ensue. Sometimes when intermittent fluctuations of intracranial pressure occur, transient blackouts may be premonitory signals of increasing intracranial pressure.

In cerebral lesions the papilledema may be greater on the side of the lesion, and papilledema does not always occur. In lesions of a frontal lobe near the optic nerve, direct pressure may be exerted upon it, giving a primary type of atrophy with pallor, while papilledema may be present in the opposite nerve head from the general rise of intracranial pressure, the Foster Kennedy syndrome. The mechanism is considered to be due to blockage of the subarachnoid space around the nerve or pressure on the central retinal vein as it crosses it. The result is swelling of neurofibrils and their eventual destruction unless intracranial pressure is relieved in time.

Papilledema generally develops in a matter of days, weeks or months, but can occur in a matter of hours. Expanding lesions in the posterior fossa do not always give papilledema; on the contrary pallor may occur.

Signs of acute increase of intracranial pressure may or may not include all or any of the above. In addition there are some signs which are very characteristic of acute compression and such occur in varying degrees. They are of very great importance in head injury and are described again under that heading.

Third nerve paralysis with progressive and rather rapid dilatation of the pupil and loss of the light reflex on the side of the lesion, often associated with some degree of extrinsic ocular paralysis, is believed to result from herniation of the hippocampal gyrus over the free edge of the tentorium which compresses the oculomotor nerve. In late cases the posterior cerebral artery may be occluded giving infarction. In addition a similar sequence of events may follow on the opposite side.

The pulse is generally slowed by acute rises of intracranial pressure and the pulse pressure widened with increase of systolic and some lowering of diastolic pressure. This is no doubt an effect on the medullary centers, as is also vomiting and hiccoughs.

Drowsiness and unconsciousness are signs of compression. Yawning is said to be a sign of a lesion within the third ventricle. Rubbing of the nose has been given a similar significance. The respiratory rhythm may be affected in increased intracranial pressure. In severe cases this may lead to Cheyne-Stokes rhythm and finally cessation of respiration. Pressure on the medulla readily causes cessation of respiration, and the slightest manipulation of the lower brain stem will cause a variation in rhythm. A rapid, stimulated, but regular respiratory rate occurs in certain types of upper brain stem or diencephalic involvement.

Herniation at the level of the incisura tentorii may cause some compression of the

cerebral peduncles against the edge of the tentorium and thus give rise to various degrees of corticospinal tract involvement including hemiparesis, spasticity, and reflex changes. In some cases the paralysis is on the ipsilateral side which is due to notching of the opposite cerebral pedicle. Frequently there is some involvement of both sides and a tendency toward decerebrate rigidity. Herniation of the cerebellar tonsils through the foramen magnum also occurs as a result of increased intracranial pressure. In the extreme this results in death as a result of involvement of the respiratory center.

Focal Signs

Focal signs must be assessed on the basis of the anatomy and the physiology of the brain. A full description of typical syndromes would require considerable space and is beyond the scope of this chapter. However, one may work out many possible combinations of symptoms and signs by taking cognizance of simple anatomical and physiological data.

Localization can generally be made upon accurate history and clinical examination. Focal signs are produced by the local compression or disturbance of the lesion. They will depend upon the physiology of the particular areas involved.

The frontal lobe is that portion of the cerebral hemisphere which lies in front of the fissure of Rolando. Lesions of the frontal lobe are apt to be associated with various mental and behavioral disturbances which may be obvious or may be so slight as to pass recognition. Lack of initiative, concentration, and impaired judgment, *laissez-faire* attitude, slovenly habits, loss of ethical standards, and moral impairment have been described. These disturbances also may accompany a temporal lobe lesion. It is thought that such symptoms are more severe when the lesion involves the dominant hemisphere.

If the lesion is large enough or if the lesion is so placed as to disturb the motor

cortex or the radiations from it, various grades of paralysis will result according to the exact position relative to the motor cortex. Thus a lesion anterior to the motor

In the dominant hemisphere motor speech seems to be subserved by an area immediately above the fissure of Sylvius and anterior to the motor cortex in the posterior

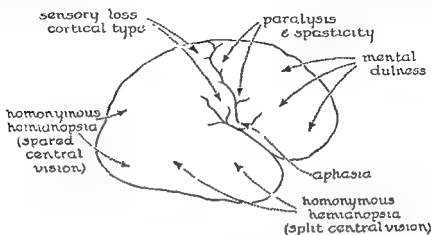


Fig. 73—Localization of certain lesions in the cerebral hemispheres which produce obvious disturbance of function. (After Penfield and McEachern)

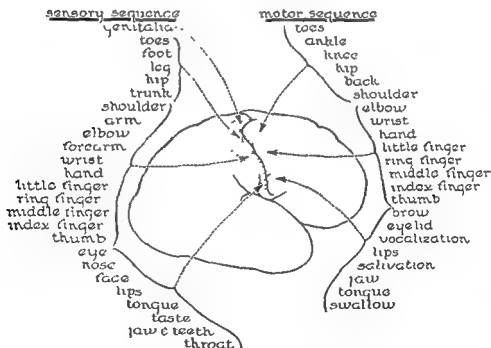


Fig. 74—Motor and sensory sequences along the fissure of Rolando. (After Penfield and Boldrey, 1937)

cortex in the parasagittal region will give maximal disturbance in the foot, whereas one in the fissure of Sylvius will give most change in the face and speech mechanism. Portions of the inferior frontal gyrus where it is subdivided by the two anterior rami of the lateral sulcus, forming the pars triangularis. A lesion in this general area will give

rise to a disturbance in speech in which the patient understands and may even know what he wants to say but cannot utter the words. When this is on an intellectual level it is called motor aphasia or expressive aphasia. This is to be differentiated from dysarthria, which consists of an inability to execute the movements necessary for normal articulation.

Generally, disturbances of speech are of a mixed type and difficult of anything but the simplest analysis. It must also be realized that much of the speech mechanism must lie beneath the cortex.

Stimulation of the brain at certain points in front of the motor cortex in the second frontal convolution provokes conjugate eye turning to the opposite side. Sometimes one encounters an irritative lesion which causes the eyes to be rotated to the opposite side and rarely a paralytic lesion which allows them to swing to the side of the lesion, presumably from relative overstimulation from the opposite side. In an epileptic seizure this is more commonly seen in the phase of exhaustion. It is known that other areas of the cortex when stimulated will cause deviation of the eyes to the opposite side. Irritability and hyperactivity are thought to occur from lesions of certain areas on the undersurface of the frontal lobe. Disturbances of autonomic function are thought to arise from lesions of the frontal lobe.

The parietal lobe is bounded in front by the fissure of Rolando and posteriorly by the parieto-occipital fissure, and below by the fissure of Sylvius. It contains the postcentral convolution which includes cell stations for discriminatory or deep cortical sensation. Lesions in the parietal lobe which encroach on or disturb this convolution cause loss of discriminatory sense from muscles and tendons and from sensory receptors in the skin, all of which can be tested practically by testing for sense of position and two point discrimination. Such other tests as for the recognition of texture and weight can be added.

The parietal lobe contains other functions which have to do with the elaboration of receptive mechanisms, but these are not well understood. Astereognosis falls into this category.

Along the upper margin of the fissure of Sylvius there are probably centers which have to do with speech, and the author feels that when this area is involved the principal disturbance is one of impaired memory for words, giving rise to degrees of anomia. In addition lack of understanding is also evident.

Disease in the region of the angular gyrus produces a disturbance of reading, alexia, and of writing, *agraphia*, which is part of the speech mechanism.

Lesions deep in this region may cause degrees of pressure upon a segment of the optic radiation. This results in a lower quadrantic field defect on the opposite side.

The occipital lobe lies posterior to the parieto-occipital fissure and contains the calcarine cortex. Lesions here produce homonymous hemianopia to the opposite side; lesions above the calcarine fissure cause a homonymous quadrantic hemianopia below and to the opposite side and lesions below the fissure quadrantic homonymous field defects above the horizontal. Lesions posterior to the lateral geniculate body may show sparing of macular or central vision.

The nondominant temporal lobe seems to be relatively silent, but on the dominant side the first temporal convolution contains certain connections for speech which is generally severely disturbed in lesions of this region. The patient can utter words and phrases, but they are meaningless and confused. This is sometimes referred to as a *jargon aphasia*. It has been discovered by Penfield "that speech is located in the temporal lobe beginning 5 cm. posterior to the tip. No speech representation is found in the first, second or third convolution anterior to this and speech representation seems to be more in the second temporal

convolution. However, it is impossible to distinguish between first and second in this area."

Many patients with cerebral disease suffer convulsive seizures. The seizure itself may be of localizing value. Thus a seizure arising in one frontal lobe may start with the head and eyes turning to the opposite side, followed by jerking of the contralateral arm and leg and loss of consciousness. Discharges further anterior can give rise to turning of the head followed by rotation of the trunk.

aura of twinkling lights may be described from a lesion in the posterior parietal region. Attacks from the occipital lobe may precipitate transient hemianopia. Dizziness is a frequent aura in lesions of the temporal lobe. Micropsia, macropsia is described and occasionally auditory hallucinations. Sometimes a past event will reappear in the patient's mind, "deja vue" phenomenon. Autonomic phenomena may be observed in lesions of the diencephalon.

Lesions in the neighborhood of the third ventricle which involve the thalami cause

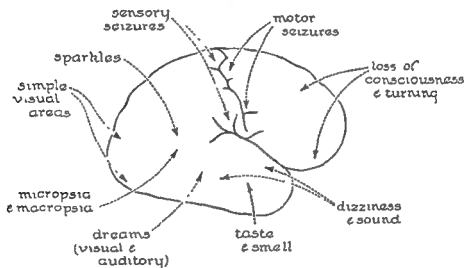


Fig 75—Common initial phenomena in epileptic seizures arising in different areas of the brain

A lesion adjacent to the motor cortex may cause clonic movements in the appropriate limb. The discharge generally spreads to neighboring areas giving rise to a Jacksonian march. Speech becomes involved if the dominant hemisphere is affected.

The accurate description of the march of events in a seizure determines its value in localization. In this the recognition of an aura is all important. Aurae may consist of a sensation rising from the epigastrium, numbness of a limb, an olfactory, gustatory, visual or auditory hallucination, according to location. For example, an attack arising in the parietal cortex, may commence with numbness of the arm, with clonic movements following as the wave passes forward. An

hypesthesia or paresthesia of the contralateral side of the body. As the internal capsule is affected there are various degrees of hemiparesis.

If the hypothalamus is involved diabetes insipidus becomes a feature. Autonomic disturbances with changes in pulse rate and blood pressure are sometimes found. Occasionally in an expanding lesion one encounters changes in the visual fields from involvement of the neighboring optic tracts. If convulsions occur they cause flushing of the face and neck, sweating, rise in blood pressure, and alteration of pulse rate.

Expanding lesions within the third ventricle cause blockage of the cerebrospinal fluid with resulting dilatation of the ven-

tricular system proximal to the lesion. There may be few objective signs except papilledema and the patient experiences severe headache which may be intermittent if the lesion is of a ball valve type.

Lesions in the midbrain give rise to various degrees of ocular paresis with pupillary changes; large pupils, sometimes small and unequal pupils, impaired reflex reactions and failure of conjugate upward eye movement may be observed. Blockage at the aqueduct of Sylvius causes internal hydrocephalus. In neoplasm of the pineal gland *pupertas praecox* is described.

Lesions of the cerebellar hemispheres generally cause incoordination of the movements of the ipsilateral extremities. Hypotonia with reduced reflexes is typical. Nystagmus is frequent in cerebellar lesions and is usually greater on gaze to the side of the lesion. Lesions of the vermis cause severe truncal ataxia and Rombergism.

Associated compression of the brain stem produces hypertonicity with increase of deep reflexes from involvement of motor tracts. Lesions of the pons and the medulla oblongata involve cranial nerve nuclei and various combinations of involvement of the long tracts. Hiccoughs, vomiting, and severe disturbances of respiration are apt to occur.

If blockade of the fourth ventricle occurs, internal hydrocephalus results with symptoms of increased intracranial pressure. Headache is severe and there may be few signs.

Tumors involving the coverings of the brain cause neurological focal signs by local compression of the brain. For example a parasagittal meningioma may compress the midline sensorimotor cortex causing characteristic signs. A meningioma of the lesser wing of the sphenoid bone causes compression atrophy of the adjacent optic nerve and, in addition, pain referred to the area supplied by the first division of the 5th cranial nerve.

An expanding lesion in the cerebellopontine angle, for example, a perineurial fibro-

blastoma of the acoustic nerve causes paralysis of the 8th, 7th and often of the 5th, and sometimes the 9th, 10th, and 11th cranial nerves as well as signs of cerebellar and brain stem involvement.

It may be added that there do seem to be sites of predilection for tumors with characteristic signs, but these are too many to list in detail.

HEAD INJURY

The subject of head injuries can be discussed under the following headings.

I. Injuries of the Brain

1. Cerebral concussion
2. Cerebral contusion
3. Cerebral laceration
4. Cerebral compression
5. Cerebral edema

II. Traumatic Intracranial Hemorrhage

1. Subarachnoid hemorrhage
2. Subdural hemorrhage
3. Epidural hemorrhage
4. Intracerebral hemorrhage
5. Subpial hemorrhage

III. Fractures of the Skull

1. Linear fracture of the vault
2. Linear fracture of the base
3. Depressed fracture, simple
4. Depressed fracture, compound

IV. Complications, Early and Late From Intracranial Injury

1. Meningitis
2. Osteomyelitis
3. Brain abscess
4. Post-traumatic headache
5. Post-traumatic dizziness
6. Post-traumatic epilepsy

Injuries of the Brain

Cerebral Concussion.—From a practical point of view cerebral concussion may be defined as a transient lapse of consciousness or memory loss following immediately upon an adequate blow to the head, and without obvious pathological findings. Because the lapse of consciousness in pure cerebral concussion is brief, it has been difficult in a clinical way to demonstrate any particular characteristic phenomena associated with it, however, recent researches have brought forth some interesting facts which give rise to further speculation.

Denny-Brown and Ritchie Russell in 1941 published an account on experimental cerebral concussion in the cat and in the macaque in which they studied the effect of concussive blows with the aid of a pendulum. They found that concussion was more easily produced when the head was free than when supported on a hard surface. From this experience they coined the terms acceleration concussion and compression concussion. They found that acceleration concussion in the monkey and cat could be produced by a heavy mass with a velocity of 28.3 feet per second and the energy transmitted 17.83 foot pounds. Following the concussive blow there was an interference with brain stem reflexes for about 90 seconds which might terminate in death or recovery within five minutes. They concluded that experimental concussion results in the following typical phenomena:

1. Loss of corneal reflex, the most important sign.
2. Loss of the pinna reflex which consists of any flicking movement of the ear, shaking of the head or scratching with the hind limb from a stimulus applied to the inner pinna.
3. A sharp rise of blood pressure, not constant.
4. Lasting inspiratory spasms only with light anesthesia.
5. An initial jerk in the musculature followed by delayed spasms, and then loss of postural tone. They are abolished by deep anesthesia.

They concluded that death from concussion results from a variety of traumatic primary shock. The primary shock is associated with and proportional to effects which suggested a stimulation of the vagoglossopharyngeal system in the medulla.

In 1944 Walker, Kollros and Case made a study of the physiological basis of concussion by the method of falling weights. They noted that concussion in the cat under Binethene, Novocain anesthesia is frequently associated with an attack, tonic and clonic.

In the monkey seizures consisted of a tonic phase and were less common. They interpret the attacks as an intense stimulation of the nervous system. Changes in respiration and circulation may occur. Usually there is a respiratory gasp followed by irregular respiration, or apnea. Usually a rise of blood pressure occurs at once or a few seconds after the blow due to an effect upon the brain stem, thus giving evidence of intense stimulation of vasomotor centers leading to peripheral vasoconstriction. They noted sometimes slowing of the pulse due to vagus excitation. Abolition of reflexes occurred only if the spinal cord was intact. These authors believed that intense stimulation of the cerebral cortex and brain stem produces excitation at the moment of the concussive blow, and that this is not due to any alteration in circulation, oxygenation, metabolism or acid base regulation of the cerebral cortex because it occurs within a fraction of a second and before such alteration can take place.

In 1946 Windle, et al., found a slight increase of water content eight to seventeen hours after experimental concussion in animals.

Electrical changes were studied in experimental concussion by Williams and Denny-Brown in 1941. They found that concussion was associated with "diminution or cessation of the electrical activity of the whole cerebral hemispheres." They observed a delayed appearance of abnormally slow waves which they felt might represent a stage in recovery from concussion. They concluded that concussion was the direct result of mechanical violence to cerebral cells and was not dependent upon secondary changes such as "edema, anoxia, hypoglycemia for its initiation."

Dow, Ulett and Raaf (1945) examined *electroencephalograms* in patients with head trauma employed in shipyards. They examined 213 cases, and in 71, *electroencephalograms* were taken within thirty to sixty minutes after the accident. They estimated

the velocity attained at the moment of impact in split seconds and determined the force involved. The percentage of abnormal records was slightly greater in patients examined within thirty minutes. Rapid disappearance of abnormal electroencephalographic findings, they felt, pointed to a mechanism in concussion other than petechial hemorrhage or contusion. The forces at impact were of the same order as those found by others to produce experimental concussion. Patients who had to stay off work more than twenty-four hours showed a greater percentage of abnormal records and a more severe injury as judged clinically, but the electroencephalographic records they found less reliable. They felt that there was a difference in susceptibility to electroencephalographic changes in different individuals on the basis of a few observations; also they did not feel that there was any particular activity that seemed characteristic of the electroencephalogram following mild trauma.

Jasper, Kershman and Elvidge (1940) found that the electroencephalogram was the most sensitive indicator of cerebral injury,

- 1 Random or regular delta waves varying in frequency from less than one to six per second.

2. Poor regulation or disorganization of the alpha rhythm

3. Epileptiform discharges.

In some cases of severe injury delta and epileptiform waves and disorganized activity were observed in the electroencephalogram several years after the injury. These were associated clinically with changes in personality, seizures, irritability, disorders of thinking and in a few instances with no remarkable clinical abnormality. Post-traumatic syndromes due to malingering or hysteria were clearly evident since in these none of the characteristic abnormalities associated with genuine head trauma were obtained.

The electroencephalogram provides an extra tool in the differential diagnosis of subdural hematoma and effusion, epidural hematoma and intracerebral hemorrhage, and focal and general trauma. According to Jasper, Kershman and Elvidge (1945), the more severe the brain injury the more complete is the disappearance of alpha rhythm and slowing of the delta frequency, and the electroencephalogram would appear to be a more sensitive measure of cerebral damage than routine clinical estimate. It was found that amnesia and confusion, irritability and drowsiness without loss of consciousness were just as important as the history of unconsciousness in indicating the severity of cerebral damage shown by the persistent electroencephalographic abnormality during the first ten days. It was found that in skull fracture severe electrical changes are more likely to be persistent and also, in cases with blood in the cerebrospinal fluid, are more likely to show persistent and severe changes, and furthermore in the presence of abnormal neurological signs immediately after the injury are associated with more severe and persistent electrical changes during the first ten days. In cases with increased cerebrospinal fluid pressure there was usually more severe abnormality. Young patients seemed to be more vulnerable from the electroencephalographic point of view. The electroencephalogram might return to normal within twenty-four hours after concussion with loss of consciousness. It was concluded, however, that records taken after the first day and up to ten days following the injury, provided a more reliable picture of residual damage to cerebral tissue and were of greater diagnostic and prognostic value. A normal or mild change in the electroencephalogram provides a useful additional guide to the management of convalescence. Local electroencephalographic abnormality may persist for years and may develop into focal epileptiform discharges associated with areas of degenerating nerve tissue. If the nervous tissue is completely destroyed the electroen-

cephalogram should be normal. Such an area may be seen, however, by pneumography. Minor epileptiform discharges may be seen without clinical seizures but may be accompanied by other symptoms. The electroencephalogram may aid in the diagnosis of intracerebral and extracerebral hemorrhage. It is not a substitute for careful clinical study of the patient but in conjunction with it makes a valuable contribution.

Cerebral Contusion.—The term cerebral contusion as used in the present instance sig-

nifies merely a bruising of the brain. This may be from the effect of the direct blow and may include so-called contre-coup lesions. Contre-coup lesions apparently occur when the skull moves when struck. According to the hypothesis of Holborn cerebral contusion and contre-coup can be explained upon the basis of rotatory acceleration. The experiments of Sheldon and Pudenz afford some evidence for this. The explanation for contre-coup still seems to be very controversial and is obviously partly a function of the inertia of the brain. Very extensive

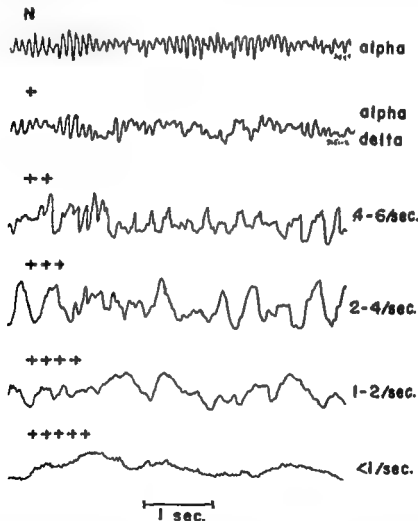


Fig. 76 —Brain waves Electroencephalogram

Typical electroencephalograms showing the changes which occur after head injury, arranged in order of severity. N is a normal record, +++++ is the most severe type of disturbance with marked depression of cortical activity. The intermediary stages are gradual and often overlap. The characteristic frequencies for each type are shown above (Jasper, Kershman, and Elvidge.)

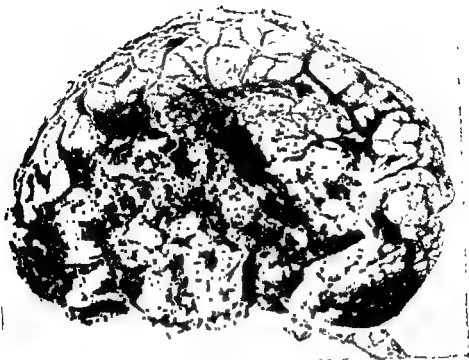


Fig. 77.—Severe contusion of the brain.



Fig. 78—Contusion or infarction of the brain stem

contusion of the brain, such as that seen in the temporal lobe, which has recently passed under the caption "explosion of the temporal lobe" (Botterell) may give rise to considerable cerebral compression due to concomitant cerebral edema.

Cerebral Laceration.—The term laceration simply means a tear in the continuity of the brain substance. This may occur as a result of a depressed fracture or as a gunshot injury and can be diagnosed only by exposure.

Cerebral Compression.—A pathological degree of cerebral compression may be brought about by intracerebral epi- and subdural hematomas, subdural effusion and massive cerebral contusion with cerebral edema. The usual signs of acute cerebral compression include increasing drowsiness, slowing of the pulse, widening of the pulse pressure, changes in respiration, progressive dilatation and fixation to light of the ipsilateral pupil and finally extensor rigidity of varying degree with plantar extension and stiff neck. Homonymous hemianopia may occur as part of the process. Hemiplegia is usually present as a focal sign of the primary lesion.

Two extremely important mechanisms operate in cases of marked increase of intracranial pressure, apart from the focal compression produced by the lesion in a particular case.

1 *Herniation of the brain at the incisura tentorii* is characterized by a herniation of the hippocampal gyrus over the free edge of the tentorium due to an ipsilateral expanding lesion. This causes pressure on the adjacent third nerve which results in various degrees of paralysis of the oculomotor nerve commencing usually with progressive enlargement of the pupil and fixation to light. Reid and Cone, 1939, found that extradural compression in monkeys would cause an ipsilateral pupillary dilatation which would return to normal on release of pressure. The fixed pupil they felt was an early sign of cerebral herniation at the incisura tentorii.

Fischer-Brügge has brought forth evidence to show that the syndrome is brought about by compression of the 3rd nerve against the edge of the clivus. Reid and Cone (1939) also confirmed the observation of Meyer that associated infarction of the ipsilateral occipital lobe might occur as a result of occlusion of the posterior cerebral artery. In severe cases the posterior cerebral artery may be compressed with resulting hemianopia. In some cases notching of the opposite cerebral peduncle against the free edge of the tentorium occurs to the extent that an ipsilateral hemiplegia is produced (Kernohan and Waltman).

2. *Herniation of the brain at the foramen magnum* consists of a forcing down of the cerebellar tonsils into the foramen magnum. This wedging causes compression of the medulla oblongata with disturbance of vital centers, causing alterations in pulse and blood pressure and early cessation of respiration. Abundance of mucus from the respiratory tubes is common.

Decerebrate rigidity with tonic seizures is due to a lesion or disturbance of the brain stem lying between the red nucleus and the acoustic striations. It may result from ischemia probably from compression of the basilar artery and its branches.

Cerebral Edema is the most feared complication in head injury and seems to be generally associated with massive cerebral contusion. If such an area of contusion consists of completely destroyed brain, and if such an area of contusion is sufficiently well localized and confined to relatively unimportant parts the best result may be obtained with a débridement of the destroyed brain. Hypertonic solutions and subtemporal decompression alone are generally completely ineffectual.

TREATMENT

Cerebral Concussion.—Modern tendency has been to treat cases of simple cerebral concussion with a minimal amount of bed rest. This will be graded according to the dura-

tion of unconsciousness and duration of confusional disturbance or memory loss. The usual case may remain in bed from two to five days. It is generally felt that the sooner the patient can be rehabilitated and sent back to work the better. His case, of course, should be properly evaluated as soon as possible after the injury, and he should be given a clear understanding as to the diagnosis and the time which he will probably be off work.

Cerebral Contusion and Laceration.—The treatment of cerebral contusion will be very much the same as that for concussion except that the more severe cases with neurological signs will require longer hospitalization. Cerebral laceration is generally combined with depressed skull fracture or gunshot injury so that treatment will be included under that section. The treatment of cerebral compression will resolve itself largely into the question of diagnosis and removal of the cause where that is possible.

SPECIAL TESTS

X-rays of the skull should be obtained in all cases of head injury. It is safer for all cases of concussion and, of course, more severe injuries, to be hospitalized, especially for the first 24 hours. The electroencephalogram is of considerable interest in the diagnosis and prognosis. Lumbar puncture is of value in demonstrating blood in the cerebrospinal fluid which gives some indication of the degree and type of injury but is of greater use in estimating increase of intracranial pressure when that is necessary.

Traumatic Intracranial Hemorrhage

Subarachnoid Hemorrhage.—This merely signifies that sufficient injury has taken place to cause bleeding into the subarachnoid space. This, in itself, may not be particularly serious. It can be diagnosed by lumbar puncture, which probably should be done in the more severe injuries in any case. This gives some idea of the severity of the injury but is only one point in the evaluation of the case. Frequent lumbar punctures

to eliminate the blood are unnecessary, and it was shown by Sprong (1934) that very little blood is removed by this method. However, less post-traumatic headache is encountered when the patient has had a combination of adequate bed rest and lumbar puncture. The general management of the case both physically and psychologically is all important.

Subdural Hemorrhage.—The diagnosis and treatment of subdural hemorrhage is often difficult though superficially it would appear simple. It must be suspected from the history, the nature of the injury and the clinical examination. Symptoms may develop rapidly with signs of increasing intracranial pressure, drowsiness, coma, stiffness of neck, hemiparesis, plantar extension, dilated pupil, pulse and blood pressure changes, or slowly, as an expanding lesion with headache. X-rays may show a shift of the pineal gland or of the choroid plexus if calcified. Ventriculography may be advisable. The final proof of course is in seeing the hematoma.

One may puncture the skull with a twist drill according to the technique of Conc. The dura can be opened simultaneously with the drill or with a needle. With a small brain needle the depth of the epidural and subdural spaces can be estimated and in many cases an epidural or a subdural hematoma recognized. If a fluid effusion is present under pressure, the measure may even be life-saving. Strategically placed burr holes through which one can inspect the subdural space may be preferable. They must be suitably placed to discover the clot. It generally lies over the convexity of the hemisphere but may lie toward the base. Usually biparietal or bitemporal openings suffice. A Penrose drain can be left in the subdural space and brought out through a separate wound. If the subdural hemorrhage is of clotted blood or of long standing with membrane formation, and especially if an inner membrane is formed, it is better policy to turn a reasonably sized bone flap and re-

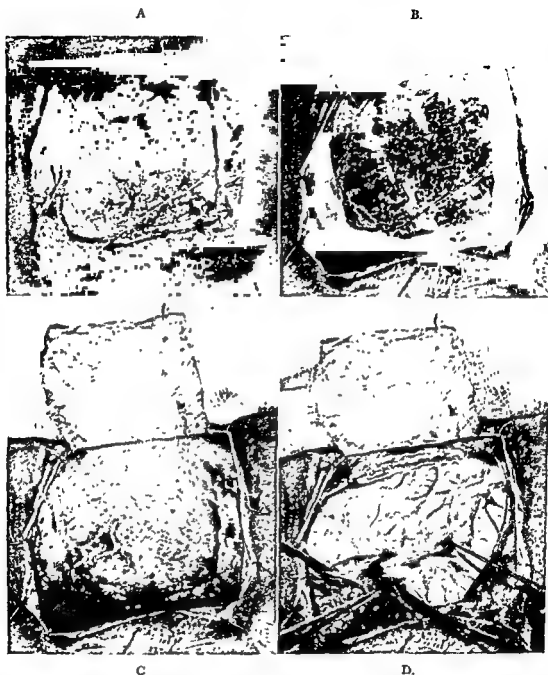


Fig 79—Case of chronic subdural hematoma, with membrane formation.

A Showing initial incision of dura, leaving partial attachments at the corners or at the crossing of the middle meningeal arteries

B Shows bulging of the outer membrane of the hematoma upon section of the dural attachment

C Reflection of dura and exposure of the outer membrane of the hematoma

D Outer membrane already partially removed and semifluid portion of the hematoma partially evacuated. Note hook lifting inner membrane away from the arachnoid. Entire membrane is removed in continuation of operation.

move the membrane completely. It is important, of course, to consider both sides in dealing with subdural hemorrhage, as frequently they are bilateral.

Epidural Hemorrhage is diagnosed again on the basis of history, examination, progress and x-ray. A head injury followed by loss of consciousness, a so-called lucid interval, and a relapse into a state of coma with paralysis and third nerve involvement is typical. An infinite number of variations occurs but the condition can usually be diagnosed

the course of the fracture if one is present. It is essential to do an immediate subtemporal craniotomy and evacuate the clot if such is suspected. If there is unnecessary delay or postponement, the brain pressure will cause death from irreparable brain stem damage.

Intracerebral Hemorrhage.—If a sizeable intracerebral hemorrhage is demonstrated, it can be removed in part by aspiration or more completely by open operation. This type of lesion is not uncommon.



Fig 80—Case of epidural hematoma after removal turned on the fracture line to permit evacuation of the clot. A small free bone flap has been removed. Note drain in lower left trephine hole. Bone has been wired back into place.

on clinical grounds alone. In some cases the acuteness of the condition will be so marked that the patient will have to be operated upon immediately in order to save his life. One can again choose the method of Cone with a twist drill to verify the diagnosis. This is somewhat quicker than waiting for x-rays, etc. This procedure is used only if one is certain that the intracranial pressure is above normal, which tends to insure against bleeding from any vessels that are encountered. When calcified, the position of the pineal gland should be observed and

Subpial Hemorrhage need hardly be mentioned but does occur and is seen during intracranial operation and occurs as a result of trauma. It has no particular significance unless it acts as an early or late irritant.

Fractures of the Skull

Linear Fracture of the Vault.—The position of the fracture and its extent will afford some idea of the type of complication to be expected. A fracture over one of the major blood sinuses may be associated with sub-

dural bleeding; if over the temporal region, with epidural hematoma. If a fracture involves the accessory air sinuses, the question of infection has to be considered. Ingress of air into the cranial cavity may even occur. The extent of fracturing gives some idea of the force of the injury which will help one in the determination of prognosis and the length of treatment. The skull should be x-rayed in all cases of head injuries.

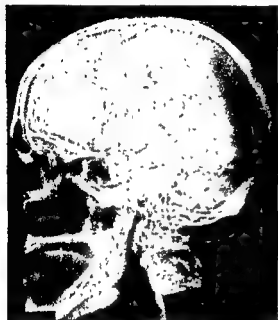


Fig 81.—X-ray of basal skull fracture showing intracranial gas

Linear Fracture of the Base.—The patient with a basal skull fracture will, generally speaking, need a longer period of bed rest.

There may be associated contusions of the brain and involvement of cranial nerves. The line of fracture frequently crosses the petrous bone to involve the mastoid air cells, with the risk of infection. Fractures often enter the frontal, ethmoidal, and sphenoidal accessory air sinuses. A leak of cerebrospinal fluid from the ear or from the nasopharynx is common. Prophylactic treatment with suitable antibiotics or chemotherapy is indicated, but meningitis may still occur. It is important to advise the patient not to sneeze forcibly or blow his nose in the presence of a cerebrospinal fluid leak from the nose. He should in that instance open his mouth widely and relax. For a leak through the ear, it is probably wisest to do no more than cover the ear with clean dressing on the outside. Most leaks stop in the first two or three days. A very free and dangerous cerebrospinal fluid leak into the nasopharynx is frequently associated with a so-called pyramidal fracture in which the facial bones have become severed from the base of the skull. This is common as a result of crashes by airplane. In this type of case one must arrange a splinting device to fix the facial bones to the skull; this can best be carried out by the oral surgeon. For certain difficult cases a method of employing a modified Roger Anderson splint applied to the skull has been described by Elvidge and Baxter (1944), which eliminates the necessity for fixation to a plaster cap.



Fig 82 —Roger Anderson splint used in case of multiple facial fractures

Depressed Fracture (Simple).—In the case of a simple depressed fracture it is routine to elevate the fragments unless the depression is very shallow and situated over a silent area of the brain. It is not urgent to reduce this in the first few hours, but it is probably wiser to elevate it within a day or two. It is advisable to make a small opening in the dura to inspect for the presence of subdural hematoma or effusion.



Fig B3.—Case of untreated compound depressed skull fracture, showing cerebral herniation covered by dirty, infected granulations and requiring prolonged period of dressings and plastic surgery

Depressed Fracture (Compound).—In this instance where the depressed fracture is covered by a laceration of the scalp, the earliest possible débridement of the wound and elevation of the depression is required.

Scalp Laceration.—It is of the utmost importance that scalp lacerations be débrided, cleansed, and properly explored lest foreign bodies or a depressed fracture be overlooked. The hair must be shaved generously about the wound, and if the wound is extensive, the whole head should be shaved in the male. The same applies even more if associated with a depressed skull fracture.

Complications, Early and Late, From Intracranial Injury

Meningitis.—Treatment is carried out with the use of suitable antibiotics and sulfa drugs. If penicillin is chosen it can be administered in six hourly doses of 300,000 units, and some combination of antibiotic therapy should be continued until one week has passed following the return of the temperature to normal. Sulfa drugs can also be administered, giving 1 Gm. of sulfadiazine every four hours after an initial dose of 2 Gm. If sulfa drugs are used, it is important to examine chemically the level which is maintained in the blood stream and also to make sure that the patient is voiding properly. The result should be followed by daily lumbar punctures. Before the introduction of penicillin and other antibiotics and of sulfa drugs, it was possible to cure some cases of streptococcal meningitis by forcing fluids and making repeated lumbar punctures every day. The treatment was called forced drainage. This type of treatment is no longer necessary. However, adequate fluid intake and lumbar punctures to check progress are essential.

Osteomyelitis.—For osteomyelitis of the skull, x-ray examination is important along with the question of tenderness and edema. The treatment is block removal of the osteomyelitic area supported by suitable antibiotics or chemotherapy.

Brain Abscess.—The treatment of brain abscess is discussed in the following section. In the final analysis it is a question of evacuation of the abscess, its proper drainage or its complete removal, with perhaps early secondary closure of the wound. If it has been removed completely and cleanly, it is possible to obtain primary closure.

It is extremely important to prevent the production of a brain abscess by adequate débridement of the original scalp laceration.

Post-Traumatic Headache.—Post-traumatic headache is part of a so-called post-concussional syndrome and is due to too long or too short a period of bed rest. It may

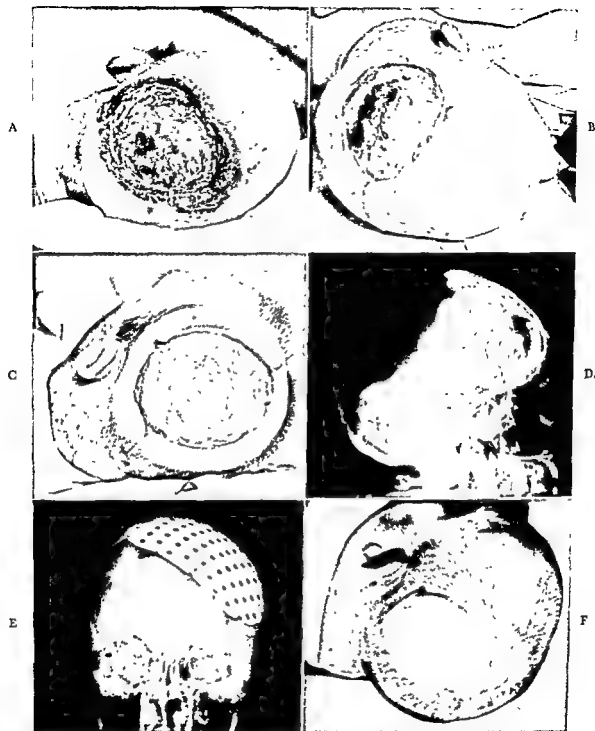


Fig 84—A Extensive electric burn which has destroyed the scalp and caused necrosis of the skull seen in the picture. The dura beneath the bone was found to have completely disappeared.

B Shows preliminary burr hole opening made through the necrotic skull and escape of large quantity of extra-arachnoidal pus.

C. Shows fascial graft to replace dura now covered with healthy red granulations about 12 days after removal of necrotic skull.

D Residual skull defect.

E. Tantalum cranioplasty.

F A pedicle flap was transferred from the abdomen to the defect in the scalp, using wrist as a carrier. (Baxter)

be due to a slight residual collection of fluid, increase of pressure, or disturbance of proper cerebrospinal fluid circulation which could have been relieved by lumbar puncture or could be on a psychological basis if the patient feels that there is something more which should have been done. It is possible in rare instances for a subdural clot or effusion to be overlooked, and in an occasional case even brain tumor. The headache may be focal in type or generalized; in some instances it follows lumbar puncture and is then due to a low intracranial pressure from a cerebrospinal fluid leak through the opening in the spinal dura.

Post-Traumatic Dizziness is described generally as an unsteady feeling which the patient states is within his own head. He notices it especially on head movement, e.g., on getting up in the morning. It is common especially when patients have not been well treated, referring especially to the minor closed head injuries. Sometimes it is associated with depressed fractures which may be associated with adhesions. Psychological adjustment in the form of simple explanation and discussion with the patient, exercise and work generally suffice to cure. If more radical measures are necessary encephalography may be advisable, which has the advantage of eliminating the possibility of a subdural hematoma and also causes sufficient shift in the pressure relationships that symptoms may disappear. It should be used with psychological reinforcement. When the dizziness due to a head injury is more vertiginous in character so that objects appear to move and to turn before the eyes of the patient, it is then probably due to a fracture involving the labyrinth. This condition clears up spontaneously in time.

Facial Paralysis.—If the 7th nerve has been paralyzed and fails to show any evidence of recovery, it indicates involvement by pressure of bony fragments within the facial canal. In some selected cases when spontaneous recovery of function does not occur, an exploratory decompression of the nerve by the otologist is advisable.

Post-Traumatic Epilepsy.—To avoid post-traumatic epilepsy, meticulous débridement should be performed at the original operation. Further than this there is little that helps. When epilepsy has developed it is a question then for evaluation and for trial on anticonvulsive therapy, and the question of surgery may be considered.

BRAIN ABSCESS

MacEwen of Glasgow obtained remarkable results by simple drainage of brain abscess. Nevertheless, since his time, results have been variable and frequently unsatisfactory for certain types of abscess. The care and skill of the surgeon has much to do with the prognosis. Much time and labor are now spared and success more apt to be assured with the antibiotics. Metastatic cerebellar abscesses have generally had a high mortality in the past. Further, the type of organism has affected the results. Modern treatment with the use of antibiotics has considerably reduced the mortality rate.

Abscesses may be divided into:

1. Single.
2. Multiple.
3. Metastatic.
4. Post-traumatic
5. Phlegmonous.

ETIOLOGY

Single abscesses most commonly arise in civilian life from mastoiditis or from frontal and ethmoidal sinusitis. From the mastoid, the abscess is generally located in the temporal lobe or the adjacent cerebellar hemisphere. From the frontoethmoidal sinuses, it is generally found in the frontal lobe.

Multiple abscesses may occur in one lobe or in scattered areas of the brain. Two abscesses have not been uncommon, one in the frontal and one in the temporal lobe, presumably from sinus infection.

Metastatic brain abscesses, which may be single or multiple, disseminate most frequently from the lungs in cases of bronchiectasis.

Post-traumatic abscess, as the name implies, is found beneath or in association with a traumatic wound of the scalp, cranium or both

Phlegmonous designates a cluster of small abscesses bound with much interstitial inflammatory tissue. This state occasionally occurs in any of the above groups

In the case of abscess from sinus infection, the process may be by direct extension through involvement of the successive coverings of the brain and spread via the Virchow-Robin spaces. This process includes focal osteomyelitis and focal pachy- and leptomeningitis. Spread may occur by retrograde venous thrombophlebitis by way of veins passings from the infected mastoid antrum, tympanic cavity or involved sinus

The infection in the metastatic type is carried by the arterial blood current as a septic embolus from a distant focus, such as a pulmonary or renal abscess. Septic emboli lodge generally in the middle cerebral distribution. Forty-five per cent of the metastatic type are solitary.

In the case of trauma, cerebral abscess formation is generally by direct extension. It may follow a simple blow but usually complicates a penetrating wound. It may come from the fracture of the base involving a sinus, or an area of softening may serve as a locus minoris resistentiae in which bacteria may lodge. The reaction of brain to infection is similar to that of other tissues. *There occurs an intense mesoblastic reaction, with exudation of leukocytes, proliferation of endothelial granulation tissue, production of macrophages from microglia and from mesothelial elements, and formation of a connective tissue capsule. The final result is a pus-filled cavity surrounded by a variably formed connective tissue capsule. The surrounding glial elements undergo hypertrophy and hyperplasia. A brain abscess from within out shows formation of a typical structure in concentric layers, with a pus-filled core surrounded by radially disposed granulation tissue and macrophages, a con-*

nective tissue capsule and with the whole surrounded by a variable thickness of reacting glial tissue

DIAGNOSIS

The general symptoms and signs are those usually seen in cases of increased intracranial pressure which may or may not be associated with pain. Focal signs may be present or absent. Convulsions may occur. A careful history will generally reveal otitis media, mastoiditis or sinusitis, a furuncle, an infected tooth, infected wound or bronchiectasis.

The story is often confusing and clinical diagnosis difficult as the following case summaries will show.

A patient who was admitted in decerebrate rigidity had suffered with his right ear since an attack of measles at the age of two years, and had undergone two operations on his right ear (mastoid) before admission. He had had a head cold a few days before with pain in the right ear, headache, drowsiness and unconsciousness on the day of admission with bilateral plantar extension, twitching of the left arm and leg and attacks of decerebrate rigidity and weakness of the left foot. The optic discs were hazy.

A patient who was found to have two abscesses suffered from a draining ear, drowsiness, and aphasia.

Another patient developed sudden headache, vomiting, dizziness, chills, prostration, normal pulse rate, stupor, coma, and aphasia. Temperature 102° to 103° F, hemiparesis, hemianopia. Diagnosis, metastatic brain abscess, probably arterial spread.

Major events taken from a more typical history can be listed briefly thus: pneumonia four months before admission, three seizures in three weeks, headache three and a half weeks, drowsiness three weeks, papilledema, and weakness of left arm recent.

Another history from a case of trauma follows: scalp laceration while tobogganing, repaired, infection, depressed fracture discovered, osteomyelitis, brain abscess drained with cure, result satisfactory but with very occasional convulsions.

The above will give sufficient idea of the variable course of the symptoms and signs in brain abscess.

Diagnosis is difficult. Accurate history of essential points, general physical and neurological examination should make one suspect the possibility of brain abscess in most cases and in many, localization can also be given.

For final diagnosis ventriculography may be used, but with an accurate clinical conclusion some would prefer to omit this. The author prefers the precision of diagnosis by ventriculography. This last is dangerous and so must be done carefully, as release of pressure places extra strain on the capsule of the abscess. Encephalography by the spinal route might be done in very simple cases, but is very dangerous on account of the possibility of cerebral herniation at the incisura tentorii or the foramen magnum.

Lumbar puncture is highly dangerous, and the leak which persists, as also the immediate drop of pressure, may cause rupture of the abscess or a cerebral herniation. The main use is to measure intracranial pressure, which, however, often gives a false reading. If checked by slight jugular compression, this adds to the risk. The principal excuse for lumbar puncture concerns the cell count in the cerebrospinal fluid, as many cases of brain abscess are associated with meningitis. The presence of both meningitis and abscess makes diagnosis very difficult. Enough has been said to show that lumbar puncture is always a hazard. It is prudent, therefore, that this be done when necessary by the man who is ready to operate.

TREATMENT

Treatment varies from time to time, and in the hands of different surgeons. The basic principle is evacuation and drainage. There are many methods, all of which must be carried out by a trained surgeon, and the postoperative treatment must be carefully supervised, otherwise re-collections are apt to occur.

Various methods of surgical treatment which have been described may be briefly listed as follows:

1. Drainage with suitable large plastic or rubber tube.
2. Aspiration of contents and insertion of suitable antibiotic on one or more occasions and if necessary subsequent drainage or surgical exposure, marsupialization or removal.



Fig 85—A Extreme ventricular displacement from case of metastatic left parietal brain abscess
B. Drainage by tube method.

3. Decompression by craniotomy with subsequent removal of the abscess as it wanders toward the brain surface and its wall thickens

4. Marsupialization of the abscess so that the abscess will eventually turn itself inside out at the surface of the brain.

5. Tube and balloon drainage.

6. Packing of the exposed cavity in an acute case with moist gauze.

7. Simple removal without rupture in a well-encapsulated case.

Simple tube drainage, though perhaps more crude than other methods, is in any event a good standard method for all types of case, and the mortality rate is very low.

Of all the methods of treatment which include the above, the author believes that simple tube drainage is the safest method in the long run and gives consistently good results. In old cases the well-formed capsule and its contents can frequently be removed without rupture. A case of metastatic abscess of the temporal lobe simulating tumor was removed in this way in toto as is customary, by the author for tumor. This proved on section to be a typhoid abscess containing motile bacilli. The patient had had typhoid fever several months before the abscess was found.

Treatment has to include rigid supervision of the postoperative dressings and control of intracranial pressure. It is important to bring the pressure low at dressings by lumbar puncture as pointed out by Cone, so as to open up the cavity. The writer prefers to keep the pressure below and to normal in the first two days. After the period of swelling is over, pressure is allowed to rise from normal to slightly above, in order to collapse the cavity more quickly. Before the era of antibiotics, the drainage tube from a cerebral abscess cavity could be removed in 12 days. Today with antibiotics it is removed in 3 to 5 days.

The mortality with the help of chemotherapy is now low if all precautions are taken. It was low before chemotherapy, but wounds

had to be more carefully supervised. The only feared complication is late epilepsy which develops in approximately one-third of the cases.

INTRACRANIAL TUMORS

Intracranial tumors may be divided into the following main groups: (after Cushing)

1. Gliomas and ganglioneuromas.
2. Meningiomas.
3. Perineurial fibroblastomas.
4. Blood vessel tumors.
5. Pituitary tumors.
6. Congenital tumors.
7. Papillomas.
8. Sarcomas.
9. Metastatic tumors.
10. Granulomas
11. Cysts

The history and the general symptoms and signs, both focal and general, produced by the majority of the above lesions, seem to become more and more characteristic for any particular tumor as one gains experience. Much can be deduced, however, from the few observations made under the preceding sections.

The symptoms and signs of disease of the central nervous system must be elucidated in terms of anatomy and physiology.

The biological behavior of the various types of tumor is exceedingly varied.

The gliomas form 44% of all brain tumors coming to a neurosurgical clinic. They arise, as the name indicates, from the neuroglia, which are the normal supportive tissue of the central nervous system. Nevertheless they are ectodermal elements. The term does not include the microglia which are thought to be, and behave physiologically as mesodermal derivatives.

To understand the classification of the gliomas, it is necessary to review the histological development of the normal glial cell which is illustrated in the accompanying diagram.

Bailey and Cushing (1926) formulated the first satisfactory classification of the gliomas basing it upon their histogenesis. Minor changes have been introduced but in the main their classification is satisfactory. As any one tumor may contain cells which are multiplying at different levels of development, one may classify a tumor according to the most primitive type of cell which is seen in any number. This is more logical as many different tumors may show a preponderance of one type of cell.

Astrocytomas may occur at any level of the central nervous system and at almost any age. The greatest incidence is in the second, third, fourth and fifth decades, with the average for the cerebral hemisphere 33.2 years, for the cerebellar hemisphere 34.2 years, for midline cerebellar cases 9.5 years, and for the spinal cord 43 years.

They are slow in growth. Preoperative symptoms average 30 months but may go on for years. Similarly the postoperative sur-

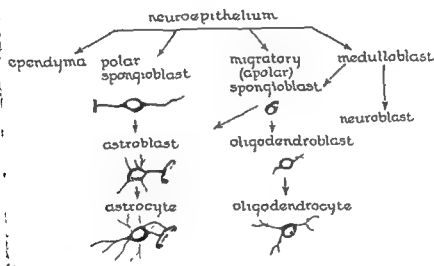


Fig 86—Development of glia. (After Penfield, 1931)

The relative incidence in a series of 210 gliomas of the central nervous system from the Montreal Neurological Institute is shown in Table VI.

TABLE VI

| | % |
|--|-------|
| Astrocytoma | 26.10 |
| Glioblastoma multiforme | 24.76 |
| Medulloblastoma | 13.33 |
| Astroblastoma | 6.19 |
| Ependymoma | 13.33 |
| Spongioblastoma polare | 5.23 |
| Oligodendroglioma | 3.80 |
| Oligodendroblastoma | |
| Pinealoma | .95 |
| Neuroepithelioma* | .47 |
| Case of astrocytoma and glioblastoma multiforme (case of triple tumor) | .47 |
| Unclassified | 9.52 |

*Includes 2 neuroepitheliomas of the peripheral nervous system.

vival may be years and there may be no recurrence.

This large group has been subclassified (of some practical importance) into at least three types:

1. Piloid
2. Gemistocytic
3. Diffusum

Into the piloid group are placed all the tumors in which the dominant cell is a fiber-forming astrocyte. It is true that there are some which are forming few fibers.

It is found that this tumor is the one which frequently forms cysts (in 3 out of 10 cerebral cases and 11 of 14 located in the cerebellum). The piloid astrocytoma may occur in the cerebrum, cerebellum, and

spinal cord, and is rather evenly distributed over six decades. It has the longest prognosis. The average age for this group when in the cerebrum is 34 years.

The *gemistocytic type* is composed of plump swollen astrocytes with a large amount of cytoplasm but few rather large, short processes and a minimum of fibers. This tumor occurs almost wholly in the cerebral hemisphere. The average age incidence is 39, and the majority occurs in the fourth and fifth decades of life. There may be a long preoperative and postoperative history, but generally less than for the piloid group. The tumor is likely to be rather fast growing. Large cysts are uncommon but medium-sized intraneoplastic cysts occur in about one-third of the cases.



Fig 87—Cystic astrocytoma showing mural nodule and ventricular shift

The *astrocytoma diffusum* is so named because of its penetrating characteristics. It is formed of small stellate astrocytes which may be called protoplasmic astrocytes. They tend to wander through the grey matter passing ganglion cells without destroying them. Sometimes groups form into more solid areas but these may not be large. Cells pass considerable distances beneath the pia. In the depth of the white matter they may infiltrate and here tend to form fibers. The average age incidence

in the above series was 34.5 years, but the majority occur toward the fifth decade in the late thirties. The preoperative symptomatology is shorter on the average but many cases go years before recognition, generally with epilepsy. Because of its infiltrating character, it is difficult to find and recognize and to delimit it at operation.

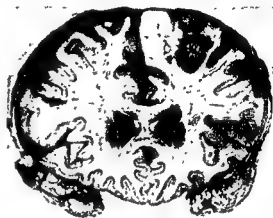


Fig 88—Gemistocytic astrocytoma showing invasion of grey and white matter and widening of convolutions



Fig 89—Astrocytoma diffusum. Shows diffuse invasion of cerebral hemisphere

The surface of the brain may look somewhat whiter than normal, but convolutions are often well preserved. X-rays show merely a uniform shift. Postmortem examination shows a general slight enlargement of the whole hemisphere. This tumor does not form cysts of any gross size and it occurs in the cerebrum. Unlike the protoplasmic astrocytoma of Bailey, it has not been found in the cerebellum. Some of these tumors can be removed *en bloc*, but sometimes it is wiser to leave them and use x-ray therapy.

The next large group of gliomas is the glioblastoma multiforme. This is a most malignant type generally found in the cere-

vessels and mitoses are frequent. Cysts may occur within the growth but are medium in size and apt to contain thick necrotic material.

There are different ways of dealing with these neoplasms. One is biopsy verification and let alone, especially if speech centers are involved. Another is radical removal. The author feels that radical removal is justified with lobectomy. One such case has now survived six years. All procedures may be followed with x-ray therapy if desired.

The third large group is the medulloblastoma group first so classified by Bailey and



Fig 90—A Arteriogram shows elevation of middle cerebral arteries by glioblastoma multiforme containing abnormal vascular network

B Shows arteriogram of the opposite normal side for comparison.

brum with an age incidence of 41.2 years and generally occurring in the fourth, fifth and sixth decades. The preoperative history is on the average about six months and frequently less. The postoperative history is short unless a very radical removal is made in which case one occasionally obtains a longer survival.

The cell type is probably the small polar spongioblast, but the tumor is generally composed of a variety of cells representing different stages of development. Malignant necrosis is common, endothelial budding of

Cushing (1925). These are primarily tumors of children in the first two decades, though curiously the average age is 19 years, as a few are seen in adults. They occur exclusively in the cerebellum, generally coming from the midline structure, the vermis, or the lateral recess of the fourth ventricle. They are highly malignant and eventually seed out into the cerebrospinal fluid and sometimes metastasize by way of cerebrospinal fluid down the spinal cord and over the brain. They have a short preoperative history, on the average 6

months, and the postoperative survival varies from a few months to several years. Radical removal followed by x-ray therapy is generally recommended. In many cases of removal recurrence has been delayed for 2 to 5 years, and rare cases are known to have survived much longer. The tumor is very sensitive to x-ray, and equally satisfactory results are reported by some where the patient is treated with x-ray therapy alone.

The remaining gliomas cannot be considered in detail here. They are less frequent. Some are relatively benign. They all have their own special biological characteristics.

The astroblastoma is somewhat more malignant than the astrocytoma. Ependymoma is moderately benign and arises in proximity to a ventricle or the neural canal. This is also typical for spongioblastoma polare. Oligodendroglioma is generally found in the cerebrum. It is rather malignant with an average age incidence of 40 years. Pinealoma is found generally in younger individuals. It may be associated with pubertas praecox.

Neuroepithelioma is highly malignant, occurs occasionally in the cerebral hemisphere, one was reported to arise from the fourth ventricle (Cushing). It has been known to arise from a peripheral nerve. Relatively good results have occasionally been obtained in cerebral cases especially in children.

One rarely encounters tumors in which the type cell belongs to the neuroblastic development. The ganglioglioma may arise at any level of the nervous system, in the cerebral hemisphere and in the cerebellum. It is to be expected in association with the spinal ganglia as a paravertebral tumor. It is relatively benign and if completely removed is not likely to recur. Neuroblastoma is composed of more immature type cells and has been found in the cerebral hemisphere and midbrain.

The meningiomas form the next major group of brain tumors (13.4% in the series

of Cushing). As the name implies, they take origin from the meninges and though generally attached to the dura, it is thought that they arise from meningeal cell rests. For example, the author has frequently noted their attachment at the site of a Pacchionian body. Different varieties have been described by Cushing, but common is the meningeal fibroblastoma. The type cell is the meningeal cell which is mesodermal and may form fibroglial fibers, reticulin and collagen.

These tumors grow to huge size before giving rise to symptoms. They merely displace brain but at their point of attachment invade dura locally and even the cranium, thickening it and passing through it. One can then see an elevation of the scalp. They arise typically at various points along the midsagittal line adjacent to the falx cerebri. They do occur along the line of the fissure of Sylvius. They commonly arise from various regions at the base; lesser wing of the sphenoid bone, dorsum sellae, tuberculum sellae, olfactory groove, floor of the middle fossa, petrous ridge, posterior fossa, foramen magnum, spinal dura and meninges.

If removed in toto including involved skull and dura if necessary, they do not recur. Sometimes they show sarcomatous tendencies in which case they may recur, even if very radically removed. They are technically difficult to remove due to general increase of vascularity of the whole region and it may be necessary to interfere with the great blood sinuses.

The perineurial fibroblastomas form another group (8.7 % in the series of Cushing). They are benign tumors which arise from perineurium and are found attached to cranial or spinal nerve roots and less often to a peripheral nerve. They do not recur when removed completely. A common type is the acoustic neuroma which occurs so frequently in connection with the 8th nerve as to derive its name from that characteristic. As the tumor enlarges in the cerebellopontine angle of the posterior fossa, it com-

presses the anterolateral aspect of the pons and the cerebellar hemisphere. Paralysis of the 8th nerve occurs very early, followed by the 7th nerve. The 5th cranial nerve is often involved and less frequently the 9th, 10th, and 11th nerves. The symptoms and

signs can be worked out on the basis of the anatomical structures affected.

Acoustic neuromas characteristically arise from the intracranial portion of the nerve at and within the entrance to the porus acousticus, which is usually seen to be en-

A.



B.



C.



D.

Fig. 91 —A Ventriculogram shows tremendous ventricular shift
 B. Shows depression of lateral ventricle and large vascular channels within the skull.
 C Arteriogram shows arterial supply to the tumor and the vascular blush revealing the growth.
 D. Vascular blush still visible in subsequent plate.

larged in x-rays. Acoustic neuromas can be removed completely, in which case they will not recur. The removal generally presupposes section of the facial nerve which leaves the patient with a disfiguring abnormality. This can be largely overcome by suitable nerve transplantation. The most satisfactory method is to join the proximal end of the 12th nerve to the distal end of the 7th, and the proximal end of the ansa hypoglossi to the distal end of the 12th cranial nerve.

The next great group are the metastatic tumors. These are generally found by the neurosurgeon in the cerebral hemisphere but may occur at any level. The usual source is from the lung.

A solitary secondary tumor of the hemisphere should be removed for relief of headache; the results are often remarkably good. The patient may recover completely for an indefinite period.

The pituitary adenomas may be divided into the chromophile or the acidophilic, the chromophobic and the basophil or basophilic, which give characteristic syndromes. They all produce certain symptoms in common. Increase of intrasellar pressure causes headache which is usually bitemporal. When the adenoma escapes from the confines of the sella turcica there is relief from headache until the local pressure rises again, or until there is an elevation in the general intracranial pressure. When the tumor is sufficiently large, pressure upon the optic chiasm results, first in an upper quadrantic bitemporal or bitemporal hemianopia. Various degrees and combinations of field defect can occasionally occur, leading eventually to blindness in one or both eyes, according to the pattern of impingement upon the chiasm and the optic nerves.

Chromophile adenomas are composed mainly of acidophilic or eosinophilic cells containing alpha granules. This tumor enlarges the sella turcica as it grows. It causes disturbances of growth hormones which lead to gigantism and acromegaly.

Chromophobe adenomas are the most common type. The cells resemble the chromophobe cells of the pituitary. These contain few if any acidophile particles. They grow to a large size and soon rupture through the confines of the sella turcica. They tend to produce symptoms of hypopituitarism.

Basophil adenomas are formed by growth of the basophilic cells of the pituitary which contain specially staining beta granules. These tumors are generally very small and can be seen on cross section of the pituitary gland. They are associated, however, with very grave symptoms known as the basophilism characterized by polycythemia, hypertension, adiposity with pendulous abdomen and striae, hypertrichosis, amenorrhea.

Adenocarcinoma of the pituitary is a rare growth; biopsy and attempted removal are made.

Epitheliomas of the hypophyseal duct arise from cell rests along the tract of the primitive craniopharyngeal duct or hypophyseal duct. They are composed primarily of epithelial cells in various types of formation with cystic spaces large or small which contain cholesterol crystals. There is usually a deposition of calcium and the growths are often seen by x-ray. The symptoms are caused primarily from a disturbance of pituitary function and pressure upon the optic chiasm and hypothalamus. These consist of disturbances of growth and nutrition and metabolism, hypophyseal cachexia or Simmond's disease, dwarfism, sexual infantilism, adiposogenital dystrophy, diabetes insipidus, failure of temperature regulation, somnolence and disturbances of vision. They are slow in growth. Removal is very difficult and frequently one has to be satisfied with mere emptying of a cyst or with partial removal.

When adenomas of the pituitary grow large enough to cause signs of compression of the optic nerves, operation is indicated.

Before this, x-ray therapy can be employed if the indications are evident. The acidophilic tumors are generally considered to be the most radiosensitive and can be held in check to a considerable degree by roentgentherapy. Hormonal therapy has to be instituted as necessary.

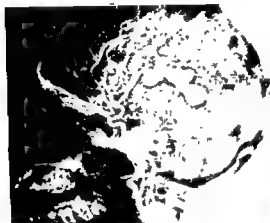
Other rare congenital tumors of the brain are cholesteatomas, dermoids, teratomas and lipomas. They occur in fairly characteristic locations and can be removed at least partially.

to cause convulsions, headache, and sometimes hemorrhage.

Papillomas occur rarely from the choroid plexus and can be removed if producing symptoms.

Sarcoma of the dura is not common. Meningiomas sometimes take on sarcomatous tendencies.

Perithelial sarcoma of the brain is not uncommon and usually occurs in the cerebral hemisphere. The treatment is removal and roentgentherapy.



A.



B.

Fig 92—A Large arteriovenous angiomatic formation fed by main branches of the middle cerebral artery.

B X-ray taken approximately 4 seconds later.

Blood vessel tumors include hemangioblastomas and hemangiomas. The former are in gross appearance not unlike a glioma but on section are found to be formed of angio-blastic or reticulo-endothelial elements. They are frequently associated with cyst and usually are located in the cerebellum. They are benign and if removed completely do not recur. Hemangiomas are difficult to differentiate from congenital anomalies of blood vessels and are probably such. The basic lesion is generally an arteriovenous connection. Cerebral arteriography is of paramount importance in the diagnosis of these lesions. Many of these formations can be removed with good results. They are prone

Melanotic sarcoma may arise from melanoblasts in the leptomeninges.

Tuberculomas can be successfully removed if they produce symptoms.

Syphilomas rarely occur in this country and are prevented now by chemotherapy.

Echinococci cysts are common in some countries; they are treated surgically.

THE VERTEBRAL COLUMN AND THE SPINAL CORD

The Vertebral Column

The vertebral column consists of thirty-three segments, 7 cervical, 12 thoracic, 5 lumbar, 5 sacral and 4 or 5 coccygeal. Both

sacral and coccygeal segments have become fused into single bony masses known as the sacrum and coccyx, respectively. The movable vertebrae are separated by the fibrocartilaginous discs. They consist of a body and a laminal arch supported by two pedicles. The apex of the laminal arch protrudes as a spinous process.

The transverse processes jut laterally as extensions of the laminal arch and pedicle. There is a superior and an inferior articular facet on either side of a vertebra which affords a certain amount of movement; this varies for each and every vertebra. The vertebrae lie in series one above the other, their flattened superior and inferior surfaces separated by discs of fibrocartilage which allow of some movement and also afford a resilient cushioning effect. The thoracic vertebrae, in addition, possess one or two facets for the head of the corresponding rib.

The vertebral bodies are held together by the strong annulus fibrosus of each disc and by the anterior and posterior spinal ligaments. Further the vertebral spines are joined by strong interspinous and supraspinous ligaments. The laminal arches are bound together by the elastic ligamenta flava.

When viewed from the side, the vertebral column exhibits a double S-shaped curve which is convex forward in the cervical and again in the lumbar regions and is concave forward in the dorsal and in the sacral regions.

The cervical vertebrae are the smallest and most movable. The upper two, the atlas and the axis, have a very special articular connection with the base of the skull and with each other which permits the necessary movements of rotation in the transverse and the anteroposterior directions. Between the atlas and the axis, besides the arthrodial diarthrosis, between the vertebral arch on each side, there is a special rotatory diarthrosis between the dens of the axis and the articular facet on the posterior surface of

the anterior arch of the atlas. Several special ligaments bind the vertebrae together.

As the cervical vertebrae possess the greatest mobility, dislocation most readily occurs in this region. In the dorsal region there is greater rigidity as it is supported by the thoracic cage. In the lumbar region there is moderate movement and a common level for fracture is at the dorsolumbar junction.

The vertebral canal lies beneath the laminal arches and the intervening ligamentum subflavum. The floor is formed by the posterior spinal ligament which lies upon the posterior aspect of the vertebral bodies and the annular capsule of the intervertebral discs. The canal is narrowest in the dorsal region.

Between each pedicle is a canal through which passes the corresponding nerve root. One root, the first, passes between the skull and the atlas. This means that the 1st cervical root passes from the vertebral canal above the first vertebra. Seven cervical vertebrae are described, but there are 8 pairs of cervical nerves, so that the 8th passes out below the 7th vertebra and the 1st thoracic nerve passes below the 1st thoracic vertebra, and all nerves below this level make their exit below the vertebra from which they are named.

The Spinal Cord

The cord is enclosed by three membranes. The inner consists of the pia mater spinalis which is said to be somewhat thicker than that which covers the brain, but the function and anatomical relationships are similar. It carries within it the blood vessels for the spinal medulla. The arachnoid is a delicate membrane continuous with that covering the brain, and between it and the pia mater is the subarachnoid space. The inner layer of the cranial *dura* extends down to form the outer tough covering of the spinal cord.

As the nerve roots leave the vertebral canal, they carry a covering of these membranes which fuses with the connective tis-

sue sheaths of the peripheral nerves on leaving the intervertebral foramina. Between each pair of nerve roots there is the dentate ligament which arises from the pia mater along the lateral side of the spinal cord. Between each root a sickle-shaped slip of this ligament passes laterally to form a pointed attachment to the dura. This occurs between each nerve down to include the 1st lumbar.

The blood supply to the spinal medulla comes from the anterior and posterior spinal arteries, and from spinal branches passing through the intervertebral foramina from the vertebral arteries and from the intercostal and the lumbar arteries. The venous drainage is by way of six longitudinal channels which form a plexus in the pia mater. This empties into the internal vertebral venous network which, in turn, empties into the vertebral, intercostal and lumbar veins and into the cranial venous sinuses and plexus above.

Until the third month of fetal life the spinal portion of the medullary tube runs the full length of the vertebral canal. From there on, however, a discrepancy in rate of growth occurs so that at birth the end of the spinal cord only reaches as far as the 1st lumbar vertebra. Because of this, nerve roots within the vertebral canal take a progressively increasing downward inclination. The lumbosacral nerves tail off below the conus of the spinal cord in what is known as the cauda equina. Puncture of the spinal subarachnoid space may be safely performed in the lumbar region below the level of the spinal cord.

In considering spinal localization, the relative position of the nerve segments and the spinal roots in relation to the vertebral column is of greatest importance. Thus the 5th lumbar nerve segment in the spinal cord lies at the level of the body of the 12th dorsal vertebra, but the nerve root runs all the way down to pass out of the vertebral canal between the 5th lumbar and the 1st sacral vertebra. A smaller discrepancy will occur

as one comes to each succeeding higher level. The 1st lumbar nerve segment probably lies at the level of the body of the 10th dorsal vertebra; the 8th dorsal over the 6th dorsal vertebra; the 1st dorsal over the 6th cervical; the 4th cervical beneath the 3rd and 4th cervical and so on. It must also be remembered that the vertebral spines in the dorsal region overlap considerably.

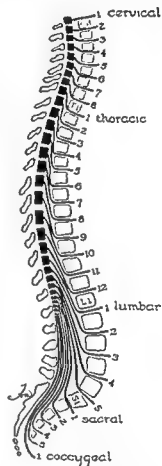


Fig 93.—Relative position of spinal nerve segments to vertebrae.

The paired nerve roots leave the spinal cord at regular intervals. Each pair of nerves which includes sensory and motor nerves, in addition to autonomic fibers in certain regions, supplies a certain area of skin and muscle. Such a region of supply is known as a spinal dermatome or nerve segment. The segmental innervation of muscles is recorded in standard textbooks of anatomy.

The cutaneous areas supplied by each nerve segment have been mapped in considerable detail in man by Head and by Foerster; in the monkey by Sherrington who employed the method of "remaining sensibility" in which three consecutive nerve roots above and three below a single intact root are sectioned. The area supplied by the remaining root can then be outlined and is the maximal for the intact root. The seg-

also for the appreciation of hot and cold sensations which are conducted in separate though closely packed laminae. Light touch is thought to pass largely by way of the contralateral ventral spinothalamic tract. Certain grades of touch also pass by the posterior columns.

Musculotendinous sensory impulses, as tested by joint position, two-point discrimination, appreciation of weight, figure

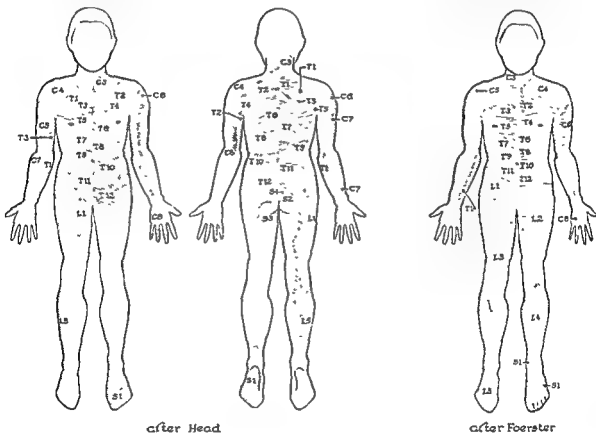


Fig. 94—Segmental dermatomal distribution

mental dermatomal distribution in man was studied in considerable detail by Head in patients with herpetic eruptions. Foerster recorded the areas of vasodilation after antidromal stimulation of the posterior roots and also mapped the dermatomal distribution by the method of remaining sensibility in clinical cases.

At this point, one may merely mention that conduction of painful sensory impulses passes up the spinal cord by way of the contralateral spinothalamic tracts. This is true

writing, vibration sense, and such discriminatory functions, pass via the posterior columns of Gall and Burdach, or the fasciculi gracilis and cuneatus.

The autonomic pathways are not well known. The lateral corticospinal tract, subserving motor impulses, lies in the lateral funiculus immediately posterior to the rubrospinal tract. Impulses to the cerebellum from muscle, tendon, and joints pass by way of the dorsal and ventral spinocerebellar tracts.

ANATOMICAL LOCALIZATION

In spinal surgery it is of first importance to know the exact level at which the lesion is situated which is determined by neurological examination.

The sensory level below which there is loss or reduction of sensation to painful stimuli may be determined. The sensation for hot and cold should be tested when more detailed examination is required; and in certain spinal lesions, a dissociation of involvement may be found. Similar testing of musculotendinous sensation must be made to determine the level of loss of function.

Autonomic disturbance may be judged by palpating the skin of the patient. The skin may be dry below the level of involvement because of the absence of sweating in the denervated areas, whereas above, it may feel moist. The presence of sweating may be demonstrated by placing the patient in a warm chamber and painting the skin with a preparation of iodine, and dusting starch powder over this. A blue coloration will occur in the presence of moisture. Other tests have been devised, and a very satisfactory method is relative measurement of skin resistance with the dermograph.

Motor power must be tested, relative weakness and paralysis listed, not forgetting the trunk musculature, for example, the intercostal muscles and the rectus abdominis. Spasticity, flaccidity and atrophy have to be adjudged. In addition the tendon reflexes, both deep and superficial, are examined. Thus, with proper examination, one may determine the level of the lesion by evaluating:

1. Sensory levels.
 - (a) pain, heat, cold, light touch
 - (b) muscle, tendon, joint sensation, i.e., position sense, two-point discrimination, figure writing, vibration sense.
2. Motor levels
 - (a) muscle power.
 - (b) muscle atrophy.
 - (c) muscle tonus.

3. Co-ordination by suitable tests.

4. Autonomic levels.

- (a) palpation of sweating level.
- (b) sweating tests, chemical.
- (c) dermograph.

5. Reflex level.

- (a) deep reflexes.
- (b) superficial reflexes.

It must be remembered that damage to the spinal cord will cause a transitory abolition of deep reflexes below the level of the lesion. The plantar reflexes may vary according to the completeness of damage. This period is known as the period of spinal shock. In the lower animals it may last but a few hours or days but in man persists for several weeks. It is said to remain longer if infection or cachexia is present.

It may be mentioned here that if the spinal cord were hemisectioned, the motor paralysis by virtue of the crossed pyramidal fibers would be ipsilateral as would be the loss of discriminatory sensation, but the loss to the modalities of pain, heat and cold would be on the opposite side due to the crossing of the fibers below the lesion. This hypothetical type of neurological manifestation is called the Brown-Séquard Syndrome. It is not usual to have such a sharply defined lesion clinically, but frequently modifications of it are seen.

In addition to the above, which may give a fairly accurate level, there are some special signs which are of great importance and afford a regional localization:

- (a) Horner's syndrome.
- (b) Paralysis of the diaphragm.
- (c) Disturbance of bladder function and weakness of the anal sphincter.

Horner's syndrome is dependent upon interruption of the autonomic sympathetic pathway at any point from the hypothalamus to the muscle controlling the pupil of the eye. The autonomic fibers pass out via the 8th cervical root and upward by way of the cervical sympathetic chain. The syndrome consists in miosis, enophthalmos, and

slight ptosis. It is most common to encounter this sign in tumors at the level of C8.

It must be remembered that the diaphragm derives its main central nerve sup-

be tested on simple palpation, and a motor level can be demonstrated, if present, in this way. The abdominal muscles can be palpated during the contraction. The um-

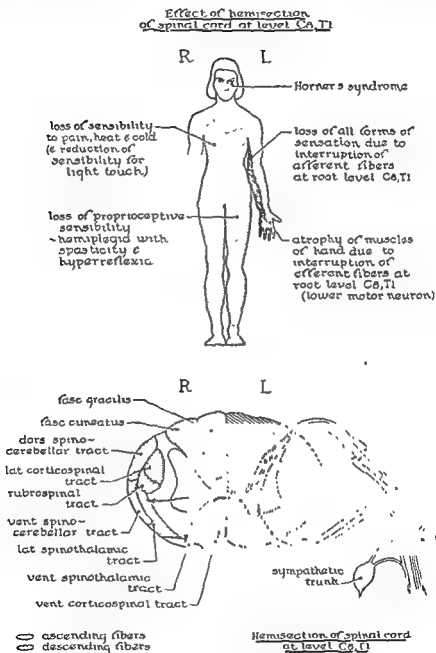


Fig. 95—Brown-Séquard Syndrome.

ply from the phrenic nerves, which emerge from or with the 3rd, 4th, and 5th cervical roots. It may also be mentioned here that the function of the intercostal muscles can

be tested on simple palpation, and a motor level can be demonstrated, if present, in this way. The abdominal muscles can be palpated during the contraction. The um-

bilicus will move upward if muscle segments below it are weakened, i.e., Beevor's sign. The bladder is supplied by sympathetic fibers from lumbar roots 1, 2, 3, (4) and by

parasympathetic fibers from roots S 2, 3, 4. Sympathetic fibers are thought to be inhibitory to the bladder musculature and evoke contraction of the internal sphincter. The parasympathetic fibers cause contraction of the detrusor muscle and relaxation of the sphincter. Cerebral autonomic fibers pass upward and downward in the spinal cord but these tracts are not as yet well-known. Normally a certain amount of voluntary control is possible. Various effects upon bladder function are produced by injury to the spinal cord. This includes a phase of shock resembling spinal shock on the somatic side in which bladder tonus may at first be lost and subsequently increased to produce a hyperactive bladder of small capacity and of frequent emptying. At this juncture, it may be well to mention that ideally bladder function in the paraplegic patient can best be treated in the initial stages, at least, by tidal drainage, which is an automatic syphonage system which rhythmically empties the bladder. After a period of treatment, it is felt that the bladder capacity will be greater than without this automatic device. In wartime it may be necessary to resort to suprapubic cystotomy.

Lumbar Puncture.—Localization of the lesion may be made very accurately by neurological examination, which can be checked by lumbar puncture studies and by myelography. One may in fact determine by spinal manometric test not only whether a sub-arachnoid block is present but also to some extent the level at which it exists. Thus on jugular compression a block in the cervical, thoracic, or lumbar region will show an absence of response in the manometer below the block. A block below the thoracic level is apt to show reduction of respiratory oscillations but fluctuations occur on pressure over the abdomen or on straining. A block at the lumbar level will show none.

In addition to the level of the lesion one may judge as to whether the lesion is intramedullary, intra- or extradural, both by the history and by the neurological examina-

tion. An intramedullary lesion is generally painless, but an extramedullary and especially an extradural one will cause pain first. There will be more gradation of sensory loss and dissociation of modality in respect of pain, temperature, and touch sensation in the intramedullary type.

The vertebral canal can be considered to be divided to some extent by the dentate ligament into anterior and posterior compartments. Some idea as to whether it lies anteriorly or posteriorly in the intervertebral canal can be adjudged by the neurological examination. Thus a spinal lesion should be described as having its upper level at such and such a spinal nerve segment, and its lower level, which is more difficult to determine, estimated.

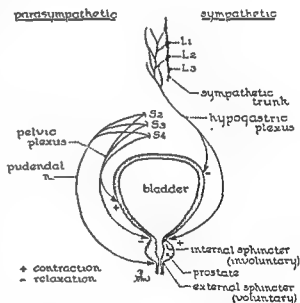


Fig. 96—Sketch of efferent nerves to the bladder.

Lumbar puncture manometric study will give evidence of absolute or relative block. This will aid in the differential diagnosis regarding presence of an expanding lesion, adhesions, etc. Due to the realization that pantopaque can be easily withdrawn at the end of the procedure, this test is generally combined or supplanted by myelography. The site of blockage can then be seen under the fluoroscope and upon x-ray films.

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It must be remembered that the diaphragm derives its main central nerve sup-

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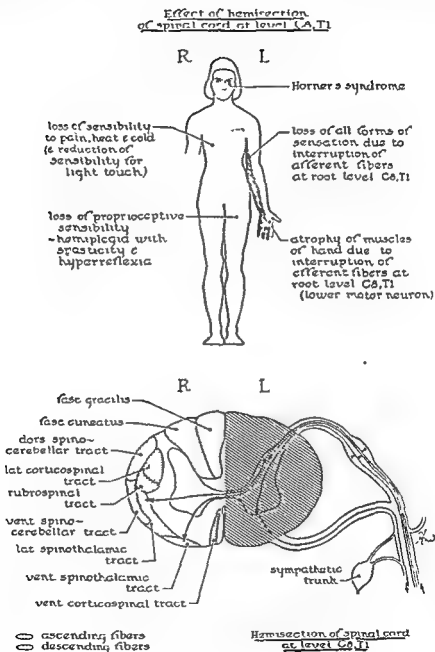


Fig 95—Brown-Séquard Syndrome.

ply from the phrenic nerves, which emerge from or with the 3rd, 4th, and 5th cervical roots. It may also be mentioned here that the function of the intercostal muscles can

blicus will move upward if muscle segments below it are weakened, i.e., Beevor's sign.

The bladder is supplied by sympathetic fibers from lumbar roots 1, 2, 3, (4) and by

In the other extreme, very simple defects may occur through the laminal arch and in rare cases are hardly more than a notching of the laminal arch. In these there is generally a thin stalk which runs out from the level of the dura into the subcutaneous tissues. The end of this is often enveloped in a ball of fatty and fibrous tissue which bulges beneath normal-appearing skin. This type is called *spina bifida occulta*.



Fig 98—Case of *spina bifida occulta* showing subcutaneous pad of fat and scar tissue dissected free, revealing stalk which enters vertebral canal.

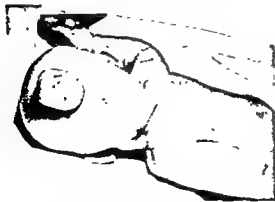


Fig 99—Cranium bifidum with meningocele.

Between these extreme examples are the meningocele and the myelomeningocele above described.

In rare cases there is an epithelial tract which runs from the skin surface to and into the dural sac and is continuous with an intraspinal epidermoid or dermoid cyst. This condition is recognizable as a button of thickened, discolored skin usually in the midlumbar region, with a small, central dimple which marks the external opening of the dermal tract. The area may be surrounded by a light growth of hair.

Spina bifida occurs occasionally in more than one member of a family and occasionally in successive generations. A family history was admitted in 6 % of cases by Ingraham, et al. Occasionally more than one meningocele presents in the same patient.

Surgical cases of *spina bifida* and *cranium bifidum* may be classified as follows:

1. *Spina bifida occulta*.
2. *Spina bifida* with meningocele.
3. *Spina bifida* with myelomeningocele or myelocele.
4. *Spina bifida* with syringomyelomeningocele.
5. *Cranium bifidum* with meningocele, encephalocele or meningoencephalocele.
6. *Spina bifida* associated with epithelial sinus and dermoid.

SYMPTOMS AND SIGNS

In the case of *spina bifida occulta* there may be no neurological defect. On the other hand there may be weakness and wasting of the muscles of one leg and sensory loss and disturbance of bladder function. The signs probably arise as a result of traction on the spinal roots or spinal cord as a result of anchorage by the *filum terminale* or nervous and fibrinous attachments. There is, of course, no emergency about operating upon this type, and patients may not be referred for treatment until adolescence when complications have become more obvious. The principal disability is loss of normal bladder function.

In the case of *spina bifida* with meningocele, myelocele, myelomeningocele and sy-

Spina Bifida and Cranium Bifidum

Lesions Arising on the Basis of a Malformation.—The neural tube closes toward the end of the third week of intrauterine life in the human fetus. Mesoblastic tissue growing between the covering ectoderm and the separated neural tube forms the membranous and bony coverings of the entire nervous system. By the 11th week the vertebral canal is covered, roofed by the neural arches from the 1st cervical to the 3rd or 4th sacral vertebral level. Occasionally there is failure in the process of closure of the laminae which results in an anomaly

the arachnoidal-like tissue is thick and spongy. With the pressure of the cerebrospinal fluid within, the meninges bulge through the skeletal defect in the form of a fluid-filled sac known as a meningocele. Often nerve roots and frequently spinal cord may be drawn into the sac which is then called a myelomeningocele. Various degrees of developmental failure of the local nervous elements are seen in these cases. In the case of herniation of brain tissue in association with cranium bifidum the anomaly is called an encephalomeningocele or encephalocele. In severe spinal defects



Fig 97—Spina bifida myelocele, showing probable attachment of cauda equina beneath center of thin dermal covering

called spina bifida. Rarely this occurs anteriorly through the body of the vertebra, usually the sacrum, in which case it is known as spina bifida anterior. When a similar failure occurs in the skull, it is known as a cranium bifidum. Experimental work, by Hertwig, produced evidence to the effect that such defects occur when an embryo is subject to abnormal environmental conditions. Mall believed that they might occur from faulty implantation as a result of endometritis or tubal pregnancy.

These bony anomalies are generally associated with failure of the meninx to differentiate into its three characteristic layers. Histologically and grossly it retains an embryonal type of structure and in some cases

there is frequently and primarily maldevelopment of the myeloid element. The neural tube itself may have failed to close in which case there is always an associated failure of the meningeal and vertebral coverings to close. Usually the laminae are widely open and the vertebral pedicles are considerably separated. The meninges are thin and the skin like transparent parchment. One can see the undeveloped neural element or primitive spinal cord running sagittally as a band along the inner side of the transparent covering. This represents the most complete type of defect, spina bifidum with myelocele, and is generally not suitable for surgery. It is sometimes called rachischisis completa.

way. Frequently one finds occipital lobe and even ventricle in the sac, and sometimes cerebellar tissue and occasionally unrecognizable tissue. Cases with moderate abnormality do well.

Cranium bifidum occurs along the midsagittal plane of the skull. It is frequent in the occipital region and rare anteriorly.

A nasopharyngeal encephalocele may occur but is rare and associated with other anomalies of the facial bones.

Sacrocccygeal teratoma and cysts of the neurenteric canal are rare but must be differentiated from meningocele. They must be removed.

Ruptured Intervertebral Disc

Protrusions of cartilaginous material into the vertebral canal have been noted from time to time by many but these were generally mistaken for tumor formations and classified as chondromata. The anatomical and pathological investigations of Schmorl (1927-31) revealed the true nature of herniations of the intervertebral discs. About this time they were recognized clinically and surgically by Stookey (1928), Dandy (1929), Alajouanine and Petit-Dutaillis (1930). Mixter and Barr (1934), demonstrated a number of cases in which sciatica was caused by herniation of the lumbar intervertebral discs. They were the first to recognize the true cause of sciatica and the general principles involved. Norlén (1944) gives credit to Antoni in 1931, for the first case diagnosed clinically.

Anatomical Considerations.—The nucleus is a semifluid plastic material enclosed by the fibrous annulus and cartilaginous plates. Owing to its physical structure, the shape and thickness of the nucleus vary with the movement of the spine and with weight-bearing. If a rupture occurs in the annulus fibrosus the nucleus escapes in part, the disc tends to collapse, and narrowing occurs between the vertebral bodies. The disc loses its semifluid consistency, and its cushioning effect is impaired.

According to Beadle (1931), osteoporosis of the spongiosa of the vertebral body results in degenerative changes, and thinning of the cartilaginous plate which allows prolapse of the nucleus pulposus into the spongiosa which has come to be known as a Schmorl's node. Symptomatically these are unimportant, but can be seen in routine x-rays and remain as tracers of degenerative changes. More important clinically are the protrusions of the intervertebral discs which occur posteriorly and posterolaterally and which cause compression of the nerve roots, and at higher levels the spinal cord. Herniations result from a constitutional idiosyncrasy or from the effects of minor traumas and associated degenerative processes.

LUMBAR DISCS

Clinical Picture.—Herniations of the disc are most likely to give rise to symptoms when they occur at the L4 to L5 and the L5 to S1 levels. This is largely due to the close proximity of the nerve root to the disc at the lower levels. They are, however, common in the cervical region at the levels C5 to C6 and C6 to C7. They least commonly give rise to symptoms in the dorsal region. When they do, they cause signs of spinal cord compression.

Patients with lower lumbar herniations generally complain of recurring attacks of low back pain and sciatica which follows the course of the affected nerve fibers. It is less common to have low back pain without sciatic radiation, and in this instance the low back pain generally spreads as far as one or the other hip. When the pain follows a sciatic distribution, it may spread according to the root involved to the ankle, the heel, the outer side, and sometimes the inner side of the foot. It may only extend to the calf or the knee, and indeed as stated may only spread toward the hip. At the L3 to L4 interspace and above, the pain is referred along the segment supplied by corresponding nerve roots.

ringomyelomeningocele, signs may range from little or no neurological involvement to the more severe degrees of sensorimotor paralysis and complete paraplegia according to the level of root and spinal cord involvement. Loss of normal function of the urinary bladder and paralysis of the anal sphincter are common. Internal hydrocephalus is a common accompaniment of this form. This is often associated with an Arnold Chiari malformation which consists of an elongation and extension of the cerebellar tonsils through the foramen magnum and various degrees of anomaly at the junction of the spinal cord and medulla. This is given as one of the causes of hydrocephalus in these cases. Other anomalies are frequently associated, e.g., clubfoot and cleft palate.

In the case of *cranium bifidum*, the neurological signs will of course depend upon the cerebral abnormality which is present. In simple cases there may be no neurological signs.

In the case of the *epithelial sinus*, there may be no neurological signs when first seen. Various degrees of paralysis are generally apparent. These are due to the intraspinal extension of the epithelial tract which is an intraspinal dermoid and behaves as an expanding growth. These cases are sometimes overlooked and are referred only when all the signs of infection have appeared with occasional septic discharge from the sinus, fever, opisthotonos, extreme pain in back and legs.

TREATMENT

This consists in plastic repair of the meningocele sac. The skin is dissected from the inner or true sac down to its base. The inner sac is then opened to explore the interior which may contain nerve elements which must be preserved. Penfield has shown that the inner sac which is composed of primitive spongy, often thick, arachnoidal-like tissue exerts an absorbing influence on the cerebrospinal fluid. For this reason he has advocated maximum preservation of this absorbing tis-

sue. Adhesions are freed as indicated and the inner sac with its nerve elements are allowed to fall free into the vertebral canal. The sac is closed appropriately and a layer of deep fascia is drawn across it. The superficial fascia and skin are then repaired.

In the average case the operation should be carried out at the age of 3 weeks when the child is best able to stand the procedure. If a clean sac is ruptured in an otherwise suitable case it should be repaired at once. If the sac is incompletely covered with epithelium and has a dirty, infected, granulosomatous surface, operation must be delayed and the sac kept clean with simple dressings until epithelization takes place and it is clean. Operations for *spina bifida occulta* can be carried out in older infants when the child is better able to stand a complicated dissection. Epithelial sinuses must be dissected carefully and removed completely along with the intradural dermoid. As these sinus tracts eventually become infected, they should be operated upon as soon as recognized. If the child is paralyzed before operation, no immediate improvement need be expected and hydrocephalus is a frequent complication.

Contraindications to operation are:

1. Complete paraplegia.
2. Marked internal hydrocephalus.
3. Complete rachischisis.
4. A dirty, infected sac.

There is little difficulty in recommending a case with simple meningocele and even myelomeningocele for operation. When there are no neurological signs, the results are excellent. However, in the face of relative paralysis or hydrocephalus, this has to be left to the judgment of the surgeon. It is generally a help to the family to have the sac repaired even in the face of paralysis. Naturally a dirty sac must be cleaned with appropriate treatment.

Cranium bifidum with meningocele and encephalocele present a similar problem, and the repair is carried out in much the same

spine following the study, and more recently by Pantopaque which is thinner and can be aspirated more easily. Up to 9 c.c. of Pantopaque is injected, and the needle remains in situ during fluoroscopy and the taking of films. At the completion of these studies the material is removed by aspiration. The material should be run as high as the thoracic region and can be carried on to the cervical if indicated.



Fig 100—A. Myelogram shows almost complete block at the level of L4 to 5 interspace.

B Shows marked spurring at the 3rd, 4th, and 5th interspaces.

C. Myelographic defect shows protrusion at level L4 to 5 on the right.

D. Myelogram shows protrusion at L5 S1 on right and evidence of bilateral protrusions at the L4 to 5 level

sidered. When they do not coincide, the question of operation has to be left to the experience of the individual surgeon. In differential diagnosis one has to consider spondylolisthesis which, of course, can be diagnosed with the x-ray and sometimes even on palpation. It may also be associated with a disc herniation. Tumor of the cauda equina, metastatic lesions, and chordoma of the vertebral column may confuse the picture. A tumor of the sciatic nerve is a rare possibility. Tuberculosis must be considered, as well as the more uncommon granulomas. It is not necessary to prolong the list of other possibilities.

Treatment.—Treatment resolves itself into nonoperative and operative. In *nonoperative* treatment bed rest alone can effect marked improvement. Plaster jacket for some six weeks, possibly in a position of slight flexion, or in any event in a comfortable position, may cause a temporary cure. It is, however, probably not much better than simple bed rest. Application of heat, massage, and possibly physiotherapy may be of value. If conservative measures fail, operative treatment may be indicated.

The question as to whether the disc should be removed, and whether simultaneous fusion should be carried out has been a source of discussion between neurosurgeons and orthopedists. In the author's opinion spinal fusion is not necessary as long as the disc has been radically removed. It is possible that some degree of soft tissue fusion will take place if the disc is radically removed, and it has been shown by Cone and Rabinovitch that when the disc is completely removed, including the cartilaginous plate in animals, actual bone fusion takes place. Because of the amount of separation between the human vertebral bodies, bony fusion in the author's opinion is not likely to occur. It is necessary to remove sufficient bone from the laminal edges for good exposure and especially from the posterior aspect of at least the entrance to the nerve root canal in order to decom-

Diagnosis.—Clinical examination and the myelogram should be in agreement, and when this is the case, operation can be con-

Generally the pain is relieved by lying down with the leg flexed. However, in some cases the pain is so severe that the patient cannot lie down and remains sitting up in bed. Standing and sitting for any length of time often give rise to more discomfort than does walking. Bending may become impossible. The act of coughing, sneezing, and straining causes sciatic radiation of pain which may be severe.

In addition to pain the patient may experience numbness and paresthesia in the distribution of a root. If a motor root is involved, weakness of the muscles supplied will result, e.g., foot drop is not uncommon in the case of a large L4 to L5 disc.

On the objective side the signs may be divided into neurological and orthopedic signs. On the neurological side one may map an area of hypesthesia over one or more root distributions of the leg and foot. Involvement of the L5 or the S1 roots is frequent, as herniations more commonly produce symptoms at these levels.

Actual muscle weakness may be present as can be demonstrated by asking the patient to pull or push against resistance while testing dorsiflexion and plantar flexion of the foot and of the toes. A useful test is to ask the patient to stand on his heels and on his toes, this will reveal weakness of the flexor muscles on the one hand and of the extensor muscles on the other. The ankle jerk is generally hypoactive or absent in an L5 to S1 herniation, but less likely to be lost in an L4 to L5 protrusion. The knee jerk is usually reduced in an L3 to L4 rupture. One may see fibrillation or fasciculation in some of the involved muscles.

The Lasègue sign is generally positive when there is clinical evidence of disc disease in the lower lumbar region. When the thigh is flexed at the hip, pain occurs when the leg is extended on the thigh, supposedly due to stretching of the sciatic nerve or a part thereof which causes radiation of pain. In some cases there occurs only pain in the lower back and hip. On the contrary,

flexion of the hip alone without extension at the knee, will not cause pain. This is helpful in making a differentiation between a spinal lesion such as a ruptured disc and a lesion about the hip joints. The sign can be intensified by dorsiflexion of the foot. In exceptional cases when the test is negative, the Lasègue sign can be modified by the raising of both legs together. This practically always gives a positive result. Tenderness over the lower lumbar spine in the gluteal region, posterior thigh, calf and the Achilles tendon can usually be elicited.

A useful test is to allow the patient to lie on his back with legs hanging over the end of the table or side of the bed. This generally causes increase of pain which can be relieved by raising one or other or both legs to the horizontal. Deviation of the legs to the right and to the left may also then accentuate the pain.

In the standing position it will generally become obvious that the patient has some degree of scoliosis, tilting of the pelvis, lumbar muscular spasm, and/or reduced lordosis and may be unable to straighten up. The patient protects the leg on the affected side by flexing the knee. In acute conditions this is sometimes quite extreme. Bending forward may increase the scoliosis, and muscular spasm, with limitation of movement which will be obvious and frequently causes kinking and locking. Certain movements produce low back pain with some degree of sciatic radiation. Bending backward and to the left and to the right will generally cause some of the above subjective and objective phenomena.

Radiological Examination.—Simple x-rays at the required level may show narrowing of the disc space with some osteophytic reaction. Increase of protein is often found in the cerebrospinal fluid. Roentgenological examination with the aid of radiopaque substances is desirable when possible. An air or an oxygen myelogram had for a time some popularity but has been superseded by Lipiodol which can be aspirated from the

of movement can be seen by positional x-rays. The diagnosis is further confirmed by myelography.

Treatment.—The results in cervical disc removal are very satisfactory. Operation in this area is more hazardous than in the lumbar region because of the proximity of the cervical spinal cord. For this reason conservative treatment should be given a very adequate trial before operative interference is considered. A collar of felt or plaster can be worn to support the chin. Bed rest for a short while and linear extension by halter traction can also be tried. It is highly important that the nerve root be adequately decompressed in the procedure of cervical discectomy which should be undertaken only by an expert in the surgery of the central nervous system. The results in this type of operation are highly satisfactory.

THORACIC DISCS

Herniations at other levels are generally nonsurgical and may fail to be recognized as they do not cause pain due to the relative position of the root in respect to the disc. However, on some occasions, they do, in which case symptoms and signs will correspond to the level involved. In the *thoracic region* they may cause compression of the spinal cord with resulting degrees of paralysis and loss of bladder function. When such is the case and when the localization is exact, operative removal is indicated. The level again can be checked by myelography. However, one must guard against artefacts of myelography especially in the thoracic region. Operation in the thoracic region is particularly difficult. Some cases of cervical and thoracic disc disease have simulated multiple sclerosis.

Fracture-Dislocation of the Spine With Neurological Involvement

The general plan of procedure has been to send the patient as rapidly as possible by ambulance or airplane to a hospital which

is sufficiently well equipped and which is best suited for dealing with these cases. This is true both in military and in civilian practice.

The diagnosis must be suspected at the time and at the site of the accident by rapid examination of the patient. The history, the clinical signs of pain or tenderness, spasm, paresthesias, anesthesia and paralysis should determine the diagnosis. If the patient is unconscious one has to rely upon the immediate history of witnesses and the objective signs. Palpation of the cervical spine may reveal loss of proper alignment; ecchymosis and swelling may be present over the dorsolumbar spine.

During transportation, if conscious, the patient must remain flat on his back for cervical fracture and his head must be braced or held between sandbags or kept in linear extension. For lumbodorsal fracture, lying prone on a stretcher may be desirable preserving normal dorsolumbar curves or supine on a firmer surface if the patient is conscious. Much will depend upon the state of respiration, as naturally a good airway is essential. The unconscious patient is transported more safely lying prone.

On arrival at hospital, neurological examination, ecchymosis, swelling, sensory level, paralysis, etc., should at once give an idea of the site of the fracturing. These will give an indication of the most important areas to be x-rayed. Distention of the bladder must be controlled by catheterization. Further treatment will depend on the type of fracturing, the neurological signs and the presence or absence of complicating factors such as multiple injuries, etc.

If *cervical fracture-dislocation* has occurred, one must consider the following surgical procedures:

Gradual reduction of the dislocation by linear traction of the spine. This can be carried out by one of three methods:

1. Halter or chin strap traction.
2. Skull traction by chrome steel wire passed through burr holes or trephine holes.

press the nerve root satisfactorily. It is important to explore adequately for the disc as on some occasions it may point more medially or more laterally, and in rare instances lie between the nerve root and the dura further caudal than one would expect, and occasionally it migrates a short distance up or down the vertebral canal.

CERVICAL DISCS

Diagnosis.—The herniated *cervical disc* presents a special problem. The signs and symptoms are similar, except for the location, to those found in the lower lumbar region giving rise to attacks of neck pain with radiation down the arm, sometimes with numbness



Fig. 101 —A Myelogram shows defect at the C6 to C7 level on the right side, suggesting large herniation of the intervertebral disc.

B X-ray shows narrowing of the intervertebral space and slight spurring at the C6 to C7 level

In carefully selected cases the results are satisfactory in at least 80 % of patients. In some the leg pain disappears while they may be left for a period of time with a feeling of weakness, stiffness, or occasional backache. This, no doubt, may be due to mechanical factors but frequently, merely to muscular spasm. It can usually be cured by suitable exercises. In the case of lumbar discectomy the author allows the patient bathroom privileges on the day following operation but otherwise prefers to keep the patient in bed for 12 to 14 days.

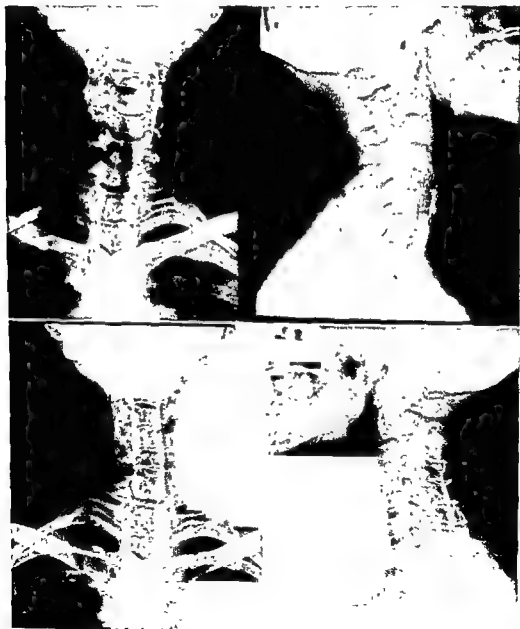
and a feeling of paresthesia and of weakness with atrophy. The most common site for the hypoesthesia is over the radial distribution, generally root C7, coming from a disc at the level of C6 to C7 and C5 to C6. Tilting the head passively to one side and jerking it slightly will frequently elicit pain in the side of the neck which may or may not radiate. There is often a point of tenderness over the transverse process of C6. X-rays of the cervical spine show a narrowing of the disc with some spurring of the bone. The normal cervical curvature is altered, and limitation

such cases cannot be properly defined in so short a space as too many variables are possible, and procedure will depend on the findings in each particular case.

Of greatest importance is the attention to the urinary bladder. This should not be allowed to become distended. Initial cath-

eterizations should be done at suitable times followed as soon as possible after operation by the establishment of an automatic bladder. It may require many weeks before this can be established. In the meantime, tidal drainage is employed, but if this is not possible (as it requires considerable care on the

A.



B

Fig. 103 —A X-rays demonstrate fracture dislocations at the level C4 to C5.

B Spinal fusion after reduction by skeletal traction of fracture dislocation at the level C4 to C5.

3. Skull traction with tongs:

- (a) Crutchfield.
- (b) Barton-Cone.

The first method is not satisfactory. It is painful and cannot be sustained long. The second is unnecessarily complicated, but is a good method. The third is simple and is satisfactory and comfortable. With these last two methods the patient can be properly nursed, turned safely, and an operation can be performed while the patient is in traction.

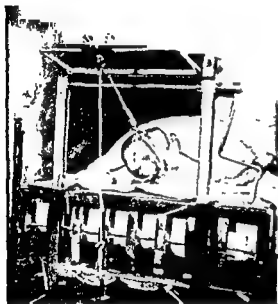


Fig 102.—Skull traction by linear extension using the Cone-Barton ice tongs

After reduction by the skull traction one must decide whether an open operation is desirable. It is preferable to explore and fuse in any case where complete dislocation has occurred or where multiple fractures have occurred usually of the body and of the laminal arches. Such an operation is made while the traction apparatus is in place. The operation consists of: (a) *Exploration*; (b) *Reduction*, which is often complete when the surgical wound is exposed. One may if necessary open the dura mater a short dis-

tance to view the condition of the cord and the vertebral canal for encroachment by bone or fibrocartilage. The dura is then repaired. One can elevate any bone which presses in from the laminal arch. When the wound is satisfactory, an internal fixation is made by wiring two or three spines together or in more complicated cases by a fusion operation usually using parallel grafts of bone from the tibia or the ribs. *Oss purum* has also been used and homologous bone from the bone bank. After the stitches are removed, a suitable plaster cast can be applied and the patient allowed up.

Similar procedures are carried out for dorsolumbar fractures, but here linear skeletal traction is difficult. In simple cases of fracturing of the body without neurological signs, the usual reduction by hyperextension can be used, but when the fracture is more complicated, includes the pedicles and arch, and especially if neurological signs are present, this maneuver is dangerous as it may cause a narrowing of the vertebral canal. In these cases, open operation, exploration, decompression, repair of dura, and replacement of roots of cauda equina, when necessary, can be carried out. This may be done with shoulder and foot traction apparatus applied during the operation and with some degree of hyperextension to correct the bony deformity. Cone has developed special apparatus for this purpose. In addition a spinal fusion is employed to hasten recovery by strengthening the spine. The operative method of treatment is preferable in most of these cases, but if this is not easily possible, the absolute indications for operation might include: a condition in which a narrowing of the vertebral canal has occurred, such as by a depression or displacement of a portion of the laminal arch or body, which has caused a degree of paralysis. Satisfactory x-rays are important in making this decision. This can be checked by lumbar puncture and a Queckenstedt test to determine whether a spinal block is present. The absolute indications to justify operation for

of the patient over the important areas. If they develop, further care is necessary. Plastic surgeons may prefer to repair with skin flaps. If this is not desirable, they can be healed by proper cleansing, débridement and nursing care.

Spasticity may become a troublesome complaint. It may be minimized by adequate physiotherapy. More radical methods of dealing with this symptom will not be discussed.

For the various grades of paraplegia the problem of late convalescence and rehabilitation has become more hopeful in the last few years due to the research of Munro, the

of Helen Barton, "he must learn to live again," so that he will become self-reliant and self-supporting.

Infection of the Spine

Tuberculous infection of the spine is more properly treated as an orthopedic condition and is discussed in that section. Only when signs of paraplegia develop from pressure of tuberculous granulation tissue or pus is the neurosurgeon involved. In such cases a decompressive laminectomy and spinal fusion are indicated.

Nontuberculous and acute pyogenic involvement of the spine may occur as an



Fig. 105—The Cone operating table with traction apparatus applied.

School for the Crippled and Disabled in New York, and of various Centers for paraplegia in Canada and the United States which have been developed since the second World War. The patient ideally should be sent to a rehabilitation center, but this is not essential. He must learn to strengthen the muscles of the shoulder girdle so that he will eventually be able to use crutches and perhaps learn to walk in splints. He can learn a gainful occupation; learn to drive an automobile; have a wheel chair, etc., and especially to look after himself, his bladder, his bowels, and to dress himself. In the words

osteomyelitis, in which case it may have arisen as a complication of a surgical procedure. Exposure and packing with antibiotics are usually necessary. In the case of spontaneous infection, it has been possible to effect a cure with antibiotics alone without operation. Rare chronic infections such as blastomycosis occasionally occur.

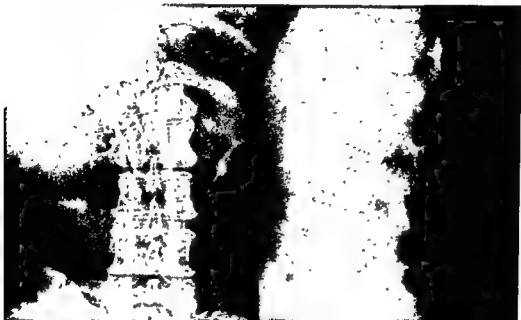
Another form of spinal infection takes place as an epidural abscess. This condition arises spontaneously apparently as a result of a metastatic infection lodging in the epidural tissues. This gives rise to general signs of acute infection to which are added spas-

part of the surgeon) one must rely upon catheterization and if necessary suprapubic cystotomy. After a certain time it should be possible to stop continuous catheterization and employ manual pressure every two or three hours at regular intervals catheterizing

for the residual. In this way the patient may develop a reflex emptying bladder. If it is not at first successful, this can be tried again after another week or so.

Of equal importance is the prevention of bed sores by sufficient turning and massage

A.



B.

Fig 104.—A. Severe fracture dislocation of the first lumbar and adjacent vertebrae.

B. Spinal fusion with parallel tibial bone grafts after exploration and decompression under skeletal traction

whiter and firmer and the nerve fibers pass into the mass rather than through its capsule. There will be no recurrence if removed completely. There may be multiple neurofibromas and other evidence of von Recklinghausen's disease.

blastoma of the cerebellum commonly metastasizes to the spinal meninges.

Syringomyelia is sometimes associated with tumor, in which case it is preferable to call it tumor with cyst, granted the cyst may traverse a considerable distance and even



Fig 106—Stages of removal in case of perineurial fibroblastoma at the level of L2. Note nerve attachment to right of retractor.

II. Tumors of the Spinal Cord.—Tumors of the spinal cord itself consist mainly of the gliomas. The more common are the astrocytomas and the ependymomas and occasionally glioblastoma multiforme and spongioblastoma polare. These tumors call for exploration. They can be removed with very satisfactory results, but in the more malignant types, and if paralysis is severe, the result will only be relative. Medullo-

the full length of the cord. Here the treatment is as for tumor. *Syringomyelia* proper occurs without tumor and is usually thought to be associated with trauma. In some cases exploration and incision of the cyst are indicated to relieve signs of pressure and possibly pain. *Syringomyelia* is associated in some cases with a congenital anomaly at the craniospinal junction in which the odontoid process is tilted backward and impinges

ticity of spinal muscles and consequently some arching of the back, together with marked tenderness and pain which may be girdlelike. Any movement causes great pain. If unrelieved, spinal compression occurs with paraplegia. The treatment is open operation, packing, and the use of antibiotics.

Tumors of the Vertebral Column

Neoplasms of the vertebral column will cause pain and eventual compression of the spinal cord. Some may be listed as follows:

A. BENIGN

- 1 Benign giant cell tumor
- Chondroma
- 3 Osteoma
- 4 Hemangioma.
5. Myeloma

B. MALIGNANT:

- Primary*—1 Osteogenic sarcoma.
2 Lymphosarcoma.

- Secondary*—1. Chordoma
2 Metastatic carcinoma.

Each cannot be discussed in detail. Suffice it to say that the ideal treatment for *benign giant cell tumor* is as complete removal as possible followed by deep x-ray therapy. In the *primary tumors*, decompressive operation, biopsy and attempted removal are desirable, followed by deep x-ray therapy.

The remaining tumors are removed as far as possible when they produce symptoms or signs.

Paget's disease is sometimes associated with sarcoma. This sometimes calls for decompression and removal. Decompression is also indicated when simple compression occurs in Paget's disease. X-ray treatment is very effective in some cases of sarcoma of the spine.

Secondary carcinoma of the spine becomes ■ neurosurgical problem when it causes pain or paraplegia. Pain can be relieved by suitable spinothalamic tract section, and para-

plegia can be prevented by decompressive laminectomy and the segmental pain can be relieved by extradural root section.

TUMORS ARISING WITHIN THE VERTEBRAL CANAL

Tumors arising within the vertebral canal may be classified in the same manner as brain tumors. They fall naturally into a group of tumors arising from the meningeal coverings of the spinal cord and the nerve roots, and another which grows from and within the spinal cord itself. It may be pointed out that there is also a group of extradural tumors but they are listed above under tumors of the vertebral column. In addition to these conditions already mentioned, Hodgkin's disease should be added; the lymphogranulomatous tissue usually lies in the epidural space with some attachment to the outer layer of the dura.

I. Tumors Arising From the Meningeal Coverings.—1. The *meningeal fibroblastoma* is a benign tumor which arises from an attachment to the dura. This neoplasm probably originates from arachnoidal cells within the dura. It is usually reddish yellow, discrete, firm, attached to the dura, finely lobulated and encapsulated. Meningiomas occur mostly in the dorsal region. This tumor must be removed completely with the involved dura. If this is done, there will seldom be a recurrence. This may be technically difficult. The author generally lays in a fascial graft or a split dural graft.

2. *Perineurial fibroblastoma* arises from the epi- and perineurium of a nerve root, usually the posterior root. These tumors are generally intradural. They are yellow, firm, discrete, benign, and must be removed completely, and the nerve root probably sacrificed. The nerve fibers spread out over the capsule of the growth and can be dissected free. If removal is complete, there will be no recurrence.

3. A *neurofibroma* has the general appearance of the above though it is somewhat

The *occipital nerves* are sometimes injected with Novocain or alcohol in cases of occipital neuralgia. Seldom is this necessary. Generally these cases are psychological. The greater occipital nerve can be injected most readily one inch lateral to the greater occipital protuberance at the level of the superior nuchal line. It can be localized more accurately by faradic stimulation.

which they supply are of importance principally in diagnosis. Thus, in a cervical fracture dislocation at the C2, C3 level one may find an anesthesia in the area supplied by the lesser occipital and the greater auricular nerves.

The most important motor branch arising in part from the level of the cervical plexus from a surgical point of view is the

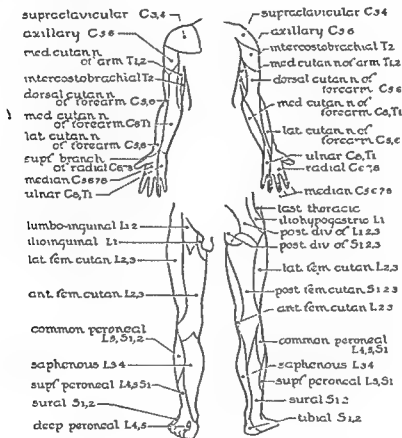


Fig 107—Peripheral nerve sensory skin areas.

Cervical Plexus.—The cervical plexus is formed by the anterior rami of the upper four cervical nerves. The sensory fibers pass by way of the lesser occipital nerves (C2, 3), great auricular nerve, nervus cutaneous colli and descending supraclavicular branches (C3, 4) to supply the skin of the lateral third of the occipital scalp, the side of the neck, and the area of skin overlying the angle of the mandible bounded above by the distribution of the trigeminal nerve. The peripheral nerve fields and the dermatomes

phrenic nerve which contains fibers from the third, fourth, and fifth anterior rami of the cervical spinal nerves. Passing obliquely across the scalenus anticus muscle the nerve must be identified and isolated in the operation for scalenotomy. Phrenicotomy with avulsion of the phrenic nerve has been used in the treatment of tuberculosis in order to collapse the lung by immobilizing the diaphragm. Regeneration seems to take place in a few months. Paravertebral growths or direct trauma may affect the phrenic nerve,

on the anterior surface of the spinal cord and medulla. Decompressive operations have been performed for this condition. In some the condition is confused with a dilated neural canal which is known as a hydromyelia of the spinal cord.

Other Rare Conditions.—Other rare conditions are lipoma of the cord which is generally irremovable without causing much disturbance of function. Spinal dermoids are difficult to remove, but with careful dissection complete removal is often possible and prognosis may be good.

Tuberculomas of the spinal cord rarely come to operation.

Adhesive arachnoiditis is a rather uncommon condition in which a dense layer of adhesions is discovered involving the pia arachnoidal membrane. Sometimes postoperative improvement results if the area of adhesion is small and delicate, arachnoiditis circumscripta, but often this process is extensive, and it is difficult to obtain regression of signs even with meticulous dissection.

PERIPHERAL NERVES

The nerve supply of the upper extremity is derived from the anterior rami of the fifth, sixth, seventh and eighth cervical and the first dorsal nerve roots. In the so-called *pre-fixed plexus* there is a contribution from the fourth cervical segment whereas in a *postfixed plexus* there is a shift caudalward in the innervation with a branch from the second dorsal root. According to Cunningham the second dorsal root always contributes a branch to the arm, the intercosto-brachial nerve with or without a connection to the brachial plexus.

The anterior spinal nerve roots are composed of the axonal processes of nerve cells situated in the anterior columns of the grey matter of the spinal cord from which they emerge as rootlets from the region of the anterolateral sulcus. The posterior spinal roots are formed by the proximal axonal processes of cells in the ganglia of the pos-

terior spinal root which lie at the level of the intervertebral foramina. The anterior and the posterior roots pierce the dura separately. Thence they share a common sheath which includes the spinal ganglion of the posterior root. The distal processes of the posterior root ganglia and of the anterior roots continue to share a common sheath and become by definition spinal nerves as they leave the intervertebral foramina.

On leaving the intervertebral foramina the spinal nerves divide into primary posterior and anterior branches. The posterior rami innervate the skin of the head and trunk posteriorly and the longitudinal muscles of the spinal axis. The anterior branches supply the whole lateral and anterior parts of the body, including the limbs, and form the great plexuses. The spinal nerves each are joined by at least one grey ramus from the paravertebral sympathetic ganglionic chain. Each anterior root, from the first dorsal to the second and third lumbar segments, supplies one white ramus communicans to the sympathetic chain with efferent fibers.

Due to regrouping in the various nerve plexuses the distribution of the peripheral nerve fields will necessarily differ from that of the sensory root dermatomes. An approximation to the general representation is given in the drawing.

Posterior Cervical Plexus.—The posterior cervical plexus as distinct from the cervical plexus is constituted by communicating branches from the posterior rami of the first, second, and the third, and possibly fourth cervical nerves. The greater occipital nerve arising from the posterior ramus of the second cervical nerve supplies sensory fibers for the posteromedial half of the occipital scalp. The third occipital nerve supplies the skin more laterally and comes from the third posterior ramus. The first cervical nerve does not generally contain sensory fibers, and motor branches are of little surgical importance.

- *2. Communicating nerve to join the phrenic nerve.
- †3. Subclavian nerve.

Posterior branches

- 1. Nerves to scalenus medius and scalenus posterior.
- *2. Dorsal scapular nerve.
- *3. Long thoracic nerve
- †4. Suprascapular nerve.

A lesion of the brachial plexus at a lower point will allow the above branches to escape. A lesion further distal toward the secondary cords will develop the aspect of a multiple peripheral nerve lesion. The lateral anterior and the medial anterior thoracic nerves, the subscapular nerves and the thoracodorsal nerve arise from the secondary cords and may be involved.

The following branches arise from the infraclavicular portion of the brachial plexus (Cunningham):

Anterior branches from the lateral cord:

- 1. Lateral anterior thoracic.
- 2. Median (lateral head).
- 3. Musculocutaneous.

Anterior branches from the medial cord:

- 1. Medial anterior thoracic
- 2. Median (medial head).
- 3. Ulnar
- 4. Medial cutaneous nerve of forearm
- 5. Medial cutaneous nerve of arm.

Posterior branches from the posterior cord:

- 1. Axillary nerve.
- 2. Radial nerve.
- 3. Two subscapular nerves.
- 4. Thoracodorsal nerve.

Lesions below the level of the secondary cords will have the characteristic distribution of peripheral nerves to the arm and forearm.

Surgical Lesions

These may be divided anatomically into those affecting

1. Spinal nerves and primary cords and branches adjacent to the vertebral column.

2. The supraclavicular portion at the point of maximal mingling in the posterior triangle of the neck.

3. The infraclavicular portion at the level of the secondary cords and branches.

Diagnosis and anatomical localization must be made by examination of the particular muscles involved and sensory loss and sympathetic nervous involvement.

Lesions may be traumatic, neoplastic, inflammatory, and trophic.

Lesions of the three primary cords or trunks may be divided into three types:

- 1. The upper cervical or the Duchenne-Erb
- 2. The middle.
- 3. The lower or the Duchenne-Aran type.

The *upper radicular* or *Duchenne-Erb* type results from a lesion of the anterior rami of the fifth and sixth cervical roots or the upper primary cord to the brachial plexus. If the damage is close to the vertebral foramina, the dorsal scapular nerve, the long thoracic nerve and branches to the scalene muscles and the longus colli may be affected. If the break occurs farther distal, these branches are spared, whereas the suprascapular nerve and the subclavian nerve may be involved together with the subscapular nerves and the lateral anterior thoracic nerves. Peripheral nerves to the arm involved may include the axillary, part of the radial, fibers from the lateral head of the median and the musculocutaneous.

Muscles in the arm and forearm which derive their total or main supply from cervical nerve roots C5 and C6 are the biceps, brachialis, brachioradialis, pronator teres, flexor carpi radialis, palmaris longus, supinator. According to level of section there may be, in addition, paralysis of the rhomboidei, levator scapulae, and serratus magnus. There may be palsy of the supraspinatus, infraspinatus, teres minor, and deltoid.

*From anterior rami of the plexus

†Level of formation of secondary cords.

6, 7, 8 and D1. Injury to the radial nerve in the arm causes paralysis of brachioradialis, triceps, extensors of wrist, fingers, and thumb. Thus paralysis is associated with wrist drop, loss of extension at the elbow, and at the metacarpophalangeal joints of the thumb and fingers. This deformity should be corrected with cock up splints. In addition a characteristic sensory loss will be observed.

It may be injured in the axilla by fractures and dislocations of the humerus, by the

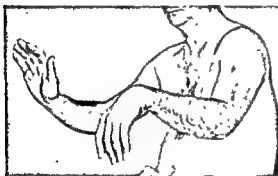


Fig 110—Wrist drop from radial nerve palsy, the result of fracture of the humerus

pressure of a crutch or by abnormal compression during sleep. It is frequently damaged in fracture of the shaft of the humerus and in gunshot wounds and other direct injuries.

Median Nerve.—The median nerve takes its origin from the fifth, sixth, seventh, and eighth cervical, and the first dorsal roots by way of the outer and the inner heads. Muscles supplied are: opponens pollicis, the outer head of the flexor pollicis brevis, abductor pollicis brevis, flexor digitorum sublimis, flexor pollicis longus, flexor carpi radialis, palmaris longus, pronator quadratus, pronator teres, lateral two lumbricals.

Disability results from involvement of flexion at wrist, loss of flexion of the thumb and index finger, paralysis of flexion at the proximal interphalangeal joints of all digits, loss of opponens action, and weakness of abduction of the thumb and of pronation of the forearm. Sensory loss occurs over palm and volar surface of thumb, index, middle finger and lateral half of the fourth fingers



Fig 111—Exposure in middle third of the arm, showing extensive thickening of radial nerve, which has been freed from dense adhesions in the neighborhood of a fracture of the humerus which had been plated. Return of function occurred in a matter of weeks. Same case as seen in Fig. 110.

The resulting deformity consists of adduction of the arm, internal rotation, extension at elbow and pronation. Conversely the patient cannot flex the arm at the elbow, supinate the forearm, externally rotate or abduct the arm. The characteristic deformity and atrophy will be diagnostic. The arm hangs with the palm of the hand rotated backward.

A lesion of the *middle primary cord* which is composed of the seventh cervical nerve causes principally extensor paralysis of the upper extremity in addition to paralysis of certain muscles about the shoulder and trunk. The deformity in the forearm is similar to that of a radial nerve lesion except that the brachioradialis may escape. Muscles involved are latissimus dorsi, subscapularis, teres major, triceps (part), the muscles supplied by the radial nerve and in addition the nerve to the coracobrachialis. The type of deformity and treatment are similar to those for the musculospiral nerve (Stookey).



Fig. 109.—Case of lower radicular palsy from traction injury. Complete paralysis of the muscles of the hand, loss of sensation along the medial side of the limb. Horner's syndrome was present.

The *lower radicular palsy* (Duchenne-Aran) occurs with damage to the eighth cervical and first thoracic nerves. This gives rise to a paralysis of muscles supplied by the ulnar nerve and the medial head of the median nerve. Muscles involved are: flexor carpi ulnaris, flexor digitorum profundus, flexor digitorum sublimis, flexor pollicis longus and the intrinsic muscles of the hand. Abductor pollicis and opponens pollicis may escape since they may receive fibers from C5, C6 roots (Stookey). The result is atrophy of the muscles along the ulnar side of the forearm and marked wasting of the small muscles of the hand. Paralysis of adduction and weakness of flexion at the wrist occur with paralysis of flexion, adduction and abduction of the fingers, extension of the distal two phalanges, opponens action and abduction of the fifth digit. There will be a cutaneous sensory loss for C8 and D1. In addition a Horner's syndrome may result from implication of the sympathetic rami.

In trauma various combinations of the above syndromes may occur. A certain lesion, however, may simulate an upper, middle, or a lower cervical lesion, a supra- or infraclavicular syndrome. In the adult civilian, involvement of the upper roots is most common in injuries which forcefully separate neck from shoulder as of a weight falling upon the shoulder thus stretching and tearing the upper roots and nerves forming the plexus. It occurs commonly from birth injury.

The lower roots are less frequently involved but may be injured at birth if the arm is delivered in a vertical position or in an adult who catches an object with his hand to break a fall. In injuries which cause stretching, nerve fibers may be torn from a distance, even from the spinal cord itself. Thus satisfactory suturing may be impossible. In wartime, injuries from missiles are common. Such may include severance, contusion and concussion of the nerves.

Radial Nerve.—The radial nerve derives its fibers from the posterior divisions of C5,

and dorsal surfaces of terminal phalanx of index and middle fingers.

Ulnar Nerve.—The ulnar nerve is derived from C8, D1 by way of the inner cord of the brachial plexus. Muscles involved include: flexor carpi ulnaris, inner mass of flexor digitorum profundus with the third and fourth lumbricals, adductor pollicis, flexor pollicis brevis (deep part), volar and dorsal interossei, the opponens and abductor digiti quinti. The result of paralysis is a flattened, narrow hand due to wasting of the small muscles with the fourth, fifth and to some extent the third fingers extended at the metacarpophalangeal joints and flexed at the proximal phalangeal joints giving rise to a "claw hand" deformity. In addition there is evidence of wasting along the ulnar side of the forearm, and the hand is in a position of radial deviation. Flexion and adduction of the wrist are weakened. Flexion at the metacarpophalangeal joints of fourth and fifth and to some extent of the third digit is lost. Flexion at the terminal phalanges and extension at the phalangeal joints of these digits is also affected. Adduction and abduction of the fingers are lost. There is inability to abduct and oppose with the fifth digit and to adduct with the thumb.

Circumflex Nerve.—The circumflex nerve arises from the fifth and sixth cervical nerves. It supplies the deltoid and the teres minor muscles, the shoulder joint with sensory fibers and the dermis over the deltoid region. It may be involved by crutch palsy and fracture-dislocation of the head of the humerus. A lesion of this nerve results in the appearance of flattening of the shoulder due to atrophy of the deltoid muscle. The peripheral disability is due to loss of abduction of the arm.

Musculocutaneous Nerve.—The musculocutaneous nerve supplies the biceps and the brachialis muscles and transmits sensory fibers to the cutaneous areas over the radial side of the forearm and hand. The nerve to the coracobrachialis muscle is generally

associated with it. Loss of flexion at the elbow is the result of involvement of this nerve. In certain positions some flexion is possible through the action of the brachioradialis. Injury may be from fracture or gunshot wound and compression.

Neuroma Formation

When a nerve has been left unsutured, the growing axonal processes from the central end cannot make contact with the lower segment and consequently grow into a fusiform swelling mixed with proliferating connective tissue from the nerve sheaths. This



Fig 113.—Neuroma in continuity. Ulnar nerve at the wrist.

is known as a *neuroma*. Some thickening of the traumatized end of the lower segment also occurs from mesodermal proliferation. When the nerve has not been anatomically severed one obtains a neuroma in continuity. The isolated processes in the peripheral segment undergo rapid degeneration leaving the sheath of Schwann intact. When the two

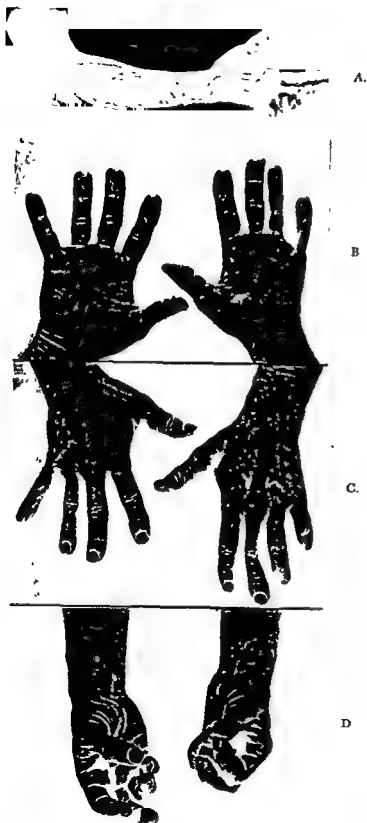


Fig. 112.—A. Case of typical deformity resulting from injury to the ulnar nerve immediately above the elbow.

B. Atrophy of the small muscles of the hand and inability to adduct or abduct the fingers

C. Atrophy and inability to extend the 4th and 5th digits at the phalangeal joints

D. Patient fails to oppose the 5th digit to the thumb

position of the nerve. The ulnar nerve may be transplanted anterior to the medial condyle of the humerus. The long end of the nerve is passed beneath the flexor digitorum sublimis and pronator teres muscles and then sutured. In making such a transplant one must consider the branches which arise at the level of transposition. In this case the branches to the flexor carpi ulnaris and to the flexor digitorum profundus may have to be sacrificed. It may be possible to bridge the gap merely by transposing the nerve anterior to the condyle and the flexor muscles. In this instance the branches to the flexor carpi ulnaris and to the flexor digitorum profundus can be spared.

Signs of returning function should be noted: return of sensation by testing light touch and pain sense; recovery of muscle tone and power by palpation, observation of the finest movements, and by disappearance of wasting. Return of function can be more precisely evaluated by the technique of electromyography when equipment is available.

Treatment of Lesions of Brachial Plexus

Traumatic lesions of the brachial plexus are difficult to treat and are apt to be discouraging in end results. Accurate diagnosis and localization are essential to satisfactory procedure. In civilian practice trauma to various parts of the brachial plexus generally occurs without open laceration. In this instance history and careful neurological examination have to be relied upon for diagnosis. It is usual to allow a certain time to elapse to decide whether regeneration is going to occur. If obvious severance has taken place, exploration and repair should be done at once. In some instances signs of recovery may appear without necessity of exploration. Nonoperative treatment consists in affording relaxation to the nerve trunks by elevation of the shoulder girdle, with flexion at the elbow. This can be accomplished by some form of airplane splint. In simple cases a sling may be suf-

ficient. In lower plexus lesions corrective splinting has to be devised. Suitable support and physiotherapy should be instituted to prevent muscular fixation at the shoulder.

In birth injury (Erb's) it is usual for recovery to take place over weeks and months without operation. But diagnosis must be accurate. Sufficient immobilization and relaxation to the shoulder must be maintained. This can be accomplished by securing the child's wrist to a soft cap or to the top of the bed.

Supraclavicular Compression Syndrome

Cervical Rib.—In order to understand this syndrome it is necessary to visualize the particular anatomical relationships that exist at the exit of the supraclavicular triangle.

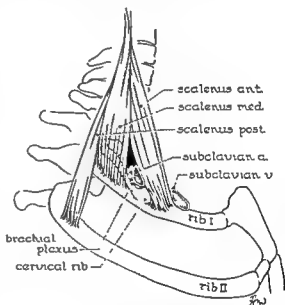


Fig 115.—Supraclavicular compression syndrome—cervical rib

Looking from the side, the anterior and the middle scalenus muscles have the shape of a tent through the door of which pass the secondary cords of the brachial plexus and the subclavian artery. The threshold is normally formed by the subclavian groove of the first rib.

The subclavian artery passes over the rib in the angle immediately behind the attach-

ends of the freshly cut nerve are brought into light apposition, the axon processes from above are directed by some chemotactic mechanism to enter the sheaths of Schwann below and down to re-innervate the part. Many fibers do not succeed in entering the degenerated nerve, grow outside it, and lose their function.

Primary Suturing of Nerves

Injury to peripheral nerves must be diagnosed through adequate history and physical findings in the light of an adequate knowledge of anatomy. In traumatic severance of a nerve, immediate suture should be done. The severed ends of the nerve may be cut clean with a very sharp blade and are laid close together in a normal position. Suturing should be carried out so that the upper point on the circumference of the cut central end corresponds as nearly as possible with the corresponding point on the upper pole of the distal segment. In this way the best possible approximation of the funiculi which may be seen within the cross section of the nerve may be hoped for. The severed ends are drawn together by interrupted sutures, which are passed through the nerve sheath.

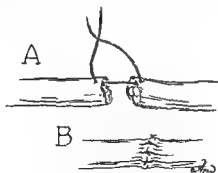


Fig 114—Nerve suture.

It is possible to rotate the nerve as necessary with these sutures. Sutures are placed at equidistant points through the sheath round the circumference of the nerve. A large nerve may require several, a small nerve two or three sutures. The two ends

of the nerve sheath are drawn together so that they are in contact. The nerve fibers under these optimal conditions should barely touch.

The suture material is very fine silk, or tantalum. Specially prepared sutures fused to needles are available. It is important to handle the nerve gently and as little as possible. Such manipulations as are necessary must be carried out by manipulating the sheath. In suturing, only the sheath should be held by the forceps. In early suture a satisfactory result can be expected. If the practitioner does not feel able to suture the nerve at the time of the accident, the wound may be débrided and allowed to heal and the patient sent along later for a secondary suture.

Secondary Suturing of Nerve

When a nerve lesion has gone unrecognized or when exploration has been deliberately delayed in order to watch for possible spontaneous recovery, which has not materialized in a reasonable time, a secondary suture can be made. This will entail excision of the neuroma and suturing. The extent of the neuroma on the proximal end and of the scarring in the distal segment may be judged by inspection and gentle palpation. If the two ends of the severed nerve are far apart it may be difficult to bridge the gap. In this instance the distance may be shortened by flexing the neighboring joint such as the elbow or the knee joint. Following suture the joint must be maintained in flexion by plaster fixation. After one to two weeks the cast may be bivalved. Passive exercise can be carried on every day and a second cast may be applied with greater extension. After three weeks it is possible for the joint to be fully extended. It is important that passive exercise be given from the start to those parts not limited by the cast, for example, to the fingers and wrist when the elbow is fixed by a cast.

A common method to effect shortening of the distance to be bridged is that of trans-

lenus anticus may be successful for all types. However, modifications are necessary in certain cases. The author has used the following techniques in the different types and advocates removal of the extra rib when present.

1. Section, scalenus anticus.
2. Section of sharp musculotendinous border of scalenus medius along with section of scalenus anticus.
3. Removal of cervical rib, including the mass at its point of junction with the first rib, beneath the artery and nerve cords, with section of the scalenus anticus.
4. Section of rudimentary band.

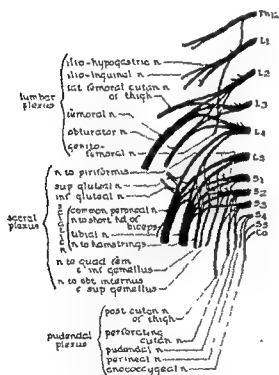


Fig. 117—Lumbar, sacral, and pudendal plexuses.

Lumbar Plexus.—The lumbar plexus is formed by the anterior branches of the first three lumbar nerves and a portion of the fourth. Occasionally there is a branch from the twelfth thoracic.

The first lumbar nerve gives rise to the ilio-hypogastric and the ilio-inguinal nerves. The genitofemoral arises from branches of

the first and second lumbar nerves. The lateral femoral cutaneous nerve of the thigh arises from the second and third lumbar nerves. Irritation of this nerve in the inguinal region from external pressure and scarring may cause pain along the anterior surface of the thigh. This condition is called *meralgia paresthetica*. It can be relieved by injection or resection of the nerve or stopping the cause.

The obturator nerve is formed from the anterior portions of the second, third, and fourth lumbar nerves. It is sometimes injured in pregnancy. It is sometimes sectioned in order to relieve adductor spasm.

The femoral nerve is formed from the posterior aspect of the second, third, and fourth lumbar nerves. This nerve supplies the quadriceps femoris, sartorius, pectineus and the iliopsoas muscles.

Paralysis causes inability to flex the thigh and to extend the knee. Sensory involvement gives loss over anteromedial aspect of thigh and leg. Particular branches supply the hip and the knee joint. The femoral nerve is rarely injured but may be involved in a pathological process especially in the paravertebral region.

The sacral plexus arises from the anterior rami of a part of the fourth lumbar nerve, the fifth lumbar, the first sacral, and portions of the second and third sacral nerves. Branches from the sacral plexus are divided into anterior (ventral) and posterior (dorsal).

They may be listed as follows (Cunningham).

Anterior branches:

1. Tibial nerve.
2. Nerves to the hamstring muscles.
3. Nerves to the quadratus femoris.
4. Nerves to the gemelli.
5. Nerves to the obturator internus.
6. Articular branches (hip).

Posterior branches:

1. Common peroneal nerve.
2. Nerves to the short head of the biceps.
3. Nerves to the piriformis muscle.

ment of the anterior scalene muscle. Arranged about its posterior, inferior, and superior surfaces are the secondary cords of the brachial plexus. The medial cord, containing the elements of the ulnar nerve and the contribution of the medial segment to the median nerve lies upon the first rib immediately behind the artery. Above it in order lie the posterior cord and the lateral cord. Immediately posterior to the cords of the plexus is the scalenus medius muscle. Normally there is just sufficient room for the roots and artery to pass, in fact, some muscular individuals are able to obliterate their pulse by contracting or tightening their scalene muscles.

In some individuals the muscles appear to be so well developed and so placed as to cause unusual compression at the exit of the supraclavicular triangle, thus causing symptoms and signs of nerve and of arterial compression. This was recognized by Adson as a syndrome many years after similar symptoms and signs were known to occur in the presence of cervical ribs. When a cervical rib is present the above portal of exit is even more crowded. A complete cervical rib will run all the way forward and will join the first rib just beneath or just behind the artery. It usually ends in a protruding bony hump which may lie beneath the artery, nerve or just behind it. This may exert just enough extra pressure to cause symptoms. In addition the extra rib tends to hold the scalenus medius rigidly forward. In this way the nerves and artery are subject to unusual compression. In incompletely formed ribs, there may be a continuing band to the first rib though this must be uncommon. In other cases where only a short rib or none is visible the tendinous band or muscular edge of the scalenus medius muscle may give pressure on the nerves.

Thus, the secondary cords of the brachial plexus may be compressed in a variety of ways each of which may produce a slightly different clinical pattern. By its musculotendinous edge the scalenus medius may play

an important part in the compression syndrome to affect principally the posterior secondary cord. On the other hand the usual ulnar involvement occurs because these fibers pass directly over the first rib or the end of the cervical rib and are more easily damaged in the above conditions.



Fig 116—Wasting of interosseous muscles. Case of cervical rib with involvement of medial secondary cord of brachial plexus

Clinical cases may be divided into groups as follows according to the manner in which the structures are affected:

1. Cases with nerve pain.
2. Cases with hypesthesia—usually ulnar, rarely radial.
3. Cases with muscular wasting and weakness, usually ulnar.
4. Cases with circulatory disturbances, even leading to gangrene.
5. Cases with autonomic disturbances, sweating, acrocyanosis.

From an anatomical point of view one might classify these cases as follows:

1. Cases in which there is no cervical rib, condition presumably due to muscular hypertrophy (sclenus anticus) or the general anatomical arrangement.
2. Cases due to compression by the musculotendinous border of the scalenus medius.
3. Cases of cervical rib complete, incomplete, and rudimentary with band.

Treatment in clearly defined examples is generally operative. Simple section of sca-

joints, and anesthesia of the skin of the lateral surface of the leg and dorsum of the foot.

In paralysis of the common peroneal nerve, homologue of the radial nerve in the arm, the knee is lifted and the foot swung forward in such a manner as to clear the ground. The patient cannot dorsiflex his foot and toes from the ground.

The tibial nerve is not frequently injured except in war casualties. Trauma results in paralysis of plantar flexion of the foot and toes, of flexion and separation of the toes, and in sensory loss over the sole of the foot and the dorsal surfaces of the terminal phalanges of the toes.

In paralysis of the tibial nerve, homologue of the median and ulnar nerves in the arm, the subject cannot raise the heel from the ground or stand on the toes.

The various lesions are again treated by primary suture when possible or by resection of the neuroma and suturing. Appliances to protect the paralyzed muscles from prolonged overstretching and the institution of properly supervised physiotherapy is of the greatest importance at all stages of treatment.

REFERENCES

- Beck, J. H.: The Cervical Spinal Cord. In: *Textbook of Neurology*, ed. 2, W. B. Saunders Company, Philadelphia, 1931, p. 161.
- Beadle, Ormond, A.: The Intervertebral Discs, Observations on Their Normal and Morbid Anatomy in Relation to Certain Spinal Deformities, Medical Research Council, London, Published by His Majesty's Stationary Office, 1931 (Special Report Series, No. 161).
- Boldrey, E. B., and Elvidge, Arthur R.: Dermoid Cysts of the Vertebral Canal, *Ann. Surg.* 110: 273-284, 1939.
- Bradford, F. Keith, and Spurling, R. Glen: The Intervertebral Disc With Special Reference to Rupture of the Annulus Fibrosus With Herniation of the Nucleus Pulposus, ed. 2, Springfield, Ill., 1945, Charles C. Thomas.
- Brock, Samuel: Injuries of the Brain and Spinal Cord and Their Coverings, ed. 3, Baltimore, 1949, The Williams & Wilkins Company.
- Brouwer, B.: Positive and Negative Aspects of Hypothalamic Disorders, *J. Neurol. Neurosurg. & Psychiat.* 13: 16-23, 1950.
- Burcy, Paul C., Heimburger, Robert F., and Oberhill, Harold R.: Compression of the Cervical Spinal Cord by Herniated Intervertebral Discs, *J. Neurosurg.* 5: 471-492, 1918.
- Camp, John D.: Contrast Myelography Past and Present. Carman Lecture, *Radiology* 54: 477-506, 1950.
- Chase, W. H.: An Anatomical Study of Subdural Hemorrhage Associated With Tentorial Splitting in the Newborn, *Surg., Gynec. & Obst.* 51: 31-41, 1930.
- Coleman, Claude C.: Peripheral Nerve Surgery—Diagnostic Considerations, *J. Neurosurg.* 1: 123-132, 1944.
- Cone, W. V., and Bridgers, Wm. H.: A Combined Tidal Irrigator and Cystometer for Management of the Paralyzed Bladder, *Surg., Gynec. & Obst.* 75: 61-66, 1942.
- Cone, W., and Turner, W. G.: The Treatment of Cervical Dislocations of the Cervical Vertebrae, *J. Neurosurg.* 1: 137, 1937.
- Craig, J. W.: Operative Rehabilitation, *J. Neurosurgery* 1: 149-155, 1944.
- Cushing, Harvey: The Pituitary Body and Its Disorders, Philadelphia, 1912, J. B. Lippincott Company.
- Cushing, Harvey: Tumors of the Nervous System and the Syndrome of the Cerebellopontine Angle, Philadelphia, 1917, W. B. Saunders Company.
- Cushing, Harvey: Studies in Intracranial Physiology and Surgery: Circulation, the Hypophysis, the Gliomas. The Cameron Prize Lectures, delivered at the University of Edinburgh, Oct. 19, 20, 22, 1925. New York, 1926, Oxford University Press.
- Cushing, Harvey: Experiences With the Cerebellar Astrocytomas. A Critical Review of Seventy-Six Cases, *Surg., Gynec. & Obst.* 52: 129-204, 1931.
- Cushing, Harvey: Intracranial Tumours: Notes Upon a Series of Two Thousand Verified Cases With Surgical-Mortality Percentages Pertaining Thereto, Springfield, Ill., 1932, Charles C. Thomas.
- Cushing, Harvey: Papers Relating to the Pituitary Body, Hypothalamus and Parasympathetic Nervous System, Springfield, Ill., 1932, Charles C. Thomas.
- Cushing, Harvey, and Eisenhardt, Louise: Meningiomas Arising From the Tuberculum Sellae With the Syndrome of Primary Optic Atrophy and Bitemporal Field Defects Combined With a Normal Sella Turcica in a Middle-aged Person, *Arch. Ophthalmol.* 1: 1-41 and 168-206, 1929.
- Cushing, Harvey, and Eisenhardt, Louise: Meningiomas: Their Classification, Regional Behaviour, Life History and Surgical End Results, Springfield, Ill., 1938, Charles C. Thomas.
- Dandy, W. E.: Loose Cartilage From Intervertebral Disk Simulating Tumor of the Spinal Cord, *Arch. Surg.* 19: 660-672, 1929.
- Dandy, W. E.: Benign Tumors in the Third Ventricle of the Brain: Diagnosis and Treatment, Springfield, Ill., 1933, Charles C. Thomas.

4. Superior gluteal nerve.
5. Inferior gluteal nerve.
6. Articular branches (knee)

The *sciatic nerve* is composed of—

1. Nerve to the hamstring muscles.
2. The tibial nerve.
3. The common peroneal nerve.
4. The nerve to the short head of the biceps muscle

The sciatic nerve is derived from the descending branch of the fourth and of the ventral and dorsal divisions of the fifth lumbar, the first, second and third sacral roots.

The *common peroneal* and the *tibial nerves* are the principal terminal branches. The common peroneal divides into three terminal branches:

1. Recurrent tibial.
2. Deep peroneal.
3. Superficial peroneal.

The first supplies the proximal fibers of *tibialis anticus* muscle, the *tibiofibular articulation* and the *knee joint*. The *deep peroneal nerve* supplies *tibialis anticus*, *extensor hallucis longus*, *extensor digitorum longus*, *peroneus tertius*, *extensor digitorum brevis*; the first, second and third *interosseous muscles*, articular branches to the ankle and the small joints of the foot, and the dorsal cutaneous area at the base of the great and second toes and their adjacent surfaces. The *superficial peroneal nerve* supplies the following muscles; *peroneus longus*, *peroneus brevis*, and the skin over the dorsum of the foot and a portion of the toes

The *tibial nerve* supplies branches to the following muscles; the two heads of *gastrocnemius*, *plantaris*, *soleus*, *popliteus*, *tibialis posterior*, *flexor digitorum longus*, *flexor hallucis longus*, *flexor digitorum brevis*, *flexor hallucis brevis*, *abductor hallucis*, *quadratus plantae*, *abductor digiti quinti*, *flexor digiti quinti brevis*, *interossei*, *adductor hallucis*, and *lumbrical muscles*. Cutaneous fibers supply the *plantar surface of the foot* and contribute the main supply to the *sural nerve* for

the *posterolateral portion* of the lower leg, *heel*, and *lateral border* of the foot, ankle, and *tarsal joints*. In addition fibers from the *tibial nerve* supply branches to the *knee joint*, the *tibiofibular joint*, and the *tarsal and metatarsal joints*.

Pudendal plexus (3rd subdivision of *lumbosacral plexus*) derives its fibers from anterior rami of first three sacral nerves and all of the anterior rami of the fourth and fifth sacral and *coccygeal nerves*. Muscles supplied by nerves from this plexus are: *levator ani*, *coccygeus*, *external sphincter*, *transversus perinei superficialis* and *profundus*, *ischiocavernosus*, *bulbocavernosus*, *sphincter urethrae membranaceae*. Cutaneous nerves supply the *perianal region*, *perineum* and *genitalia*, and the *posterior aspect* of the thigh. Autonomic fibers are dealt with elsewhere.

The *sciatic nerve* may be involved in *neoplastic growth* in the pelvis, at the *sciatic notch* and along its course. Some possibilities may be listed: *aneurysm* at the *sciatic notch*, *gravid uterus*, *sarcoma* of the upper end of the *femur* and *trauma* as in *forced manipulation* for *dislocation* of the *hip joint*, *contusion* by *fracture*, *open wounding*, and *gunshot wounds*

Complete involvement of the *sciatic nerve* causes *paralysis* of all the muscles in the leg and the foot and *partial paralysis* of the muscles of the thigh and produces *anesthesia* of the foot and the *lateral surface* of the leg. The general practical result will include a *steppage type* of gait with *complete foot drop*. Autonomic disturbances with *lack of sweating* and *edema* of the foot also may be noted.

The *common peroneal nerve* is more vulnerable than the *tibial*. In addition to the usual forms of *trauma* and *gunshot injuries*, damage occurs as a result of *pressure* on the nerve, for example, in *crossing the legs* and *pressure from casts*. It results in a *paralysis of dorsiflexion* and *eversion* of the foot, *extension* of the toes at *proximal phalangeal*

- LeBeau, J.: Metastatic Abscess of the Brain
Surgical Treatment and Results, Acta psy-
chiat. et neurol. 24: 517-558, 1919.
- Clark, W. E., LeGros, Beattie, John, Riddoch,
George, and Dott, Norman, M.: The Hypo-
thalamus: Morphological, Functional, Clin-
ical and Surgical Aspects, Edinburgh and
London, 1938, Oliver & Boyd.
- Love, J. G.: Protruded Intervertebral Disks; Re-
port of 100 Cases in Which Operation Was
Performed, J. A. M. A. 111: 396-400, 1938
- Love, J. G., and Walsh, M. N.: Intraspinal Pro-
trusions of Intervertebral Disks, Arch. Surg.
40: 454-484, 1940
- Lyons, Wm. R., and Woodhall, Barnes: Atlas of
Peripheral Nerve Injuries, Philadelphia, 1949,
W. B. Saunders Company
- MacEwen, W.: Pyogenic Infective Diseases of the
Brain and Spinal Cord, Meningitis, Abscess
of Brain, Infective Sinus Thrombosis, Glas-
gow, 1893, J. Maclehose and Sons
- Malmö, Robert B.: Psychological Aspects of
Frontal Gyrectomy and Frontal Lobotomy in
Mental Patients, A. Research Nerv. & Ment.
Dis., Proc. 27: 337-364, 1947.
- Medical Research Council: Nerve Injuries Com-
mittee. Aid to the Investigation of Periph-
eral Nerve Injuries, London, Published by
His Majesty's Stationery Office, M. R. C.
War Memorandum No 7, p 48, 1942
- Mixter, W. J., and Barr, J. S.: Rupture of the
Intervertebral Disk With Involvement of the
Spinal Canal, New England J. Med 211:
210-215, 1934
- Egas " " " " " " " " " " " " " " " "
- Egas " " " " " " " " " " " " " " " "
- Mun " " " " " " " " " " " " " " " "
- 229-239, 1935
- Norlén, Gosta: Familial Occurrence of Cerebellar
Angioma, Acta chir. Scandinav 85: 198-202,
1941
- Norlén, Gosta: On the Value of the Neurological
Symptoms in Sciatica for the Localization of
a Lumbar Disc Herniation, A Contribution to
the Problem of the Surgical Treatment of
Sciatica, Acta chir Scandinav. (suppl. 95)
91: 1-96, 1944
- Norlén, Gosta: Arteriovenous Aneurysms of the
Brain Report of Ten Cases of Total Re-
moval of the Lesion, J Neurosurg 6: 475-
494, 1949
- Penfield, W G : The Cranial Subdural Space,
Anat Rec 28: 173-175, 1924.
- Penfield, W G : Neuroglia: Normal and Patho-
logical, in Penfield, Wilder, editor Cytology
and Cellular Pathology of the Nervous Sys-
tem, New York, 1932, Paul B Hoeber, Inc.,
vol 2, section 9, pp 422-479
- Penfield, W G : Hydrocephalus and Spina Bifida,
Surg. Gynec & Obst 60: 363-369, 1935
- Penfield, W: Ferrier Lecture, Some Observations
on the Cerebral Cortex of Man, Proc Roy
Soc., London, sB 134: 329-347, 1947.
- Penfield, Wilder G : Memory Mechanisms Presi-
dential Address, 76th Annual Meeting, Am
Neurol A., Atlantic City, June 18, 1951
Arch Neurol & Psychiat. 67: 178-198, 1952
- Penfield, W., and Cone, W.: Spina Bifida and
Cranium Bifidum. Results of Plastic Re-
pair of Meningocele and Myelomeningocele
by a New Method, J. A. M. A. 98: 454-460,
1932.
- Penfield, Wilder, and Boldrey, Edwin: Somatic
Motor and Sensory Representation in the
Cerebral Cortex of Man as Studied by Elec-
trical Stimulation, Brain 60: 389-443, 1937.
- Penfield, Wilder, and Elvidge, A. R.: Hydro-
cephalus and the Atrophy of Cerebral Com-
pression, in Penfield, Wilder, editor: Cyto-
logy and Cellular Pathology of the Nervous
System, New York, 1932, Paul B Hoeber,
Inc., vol. 3, section 28, p. 1267.
- Penfield, W., and Erickson, T. C.: Epilepsy and
Cerebral Localization, Springfield, 1941,
Charles C Thomas
- Penfield, W., and Kristiansen, K.: Epileptic
Seizure Patterns, Springfield, 1951, Charles
C Thomas
- Penfield, Wilder, and McEachern, Donald: In-
tracranial Tumors, New York, 1938, Oxford
University Press, chap 6.
- Penfield, W., and Norcross, N. C.: Subdural
Traction and Post-Traumatic Headache:
Study of Pathology and Therapeutics, Arch.
Neurol & Psychiat. 36: 75-95, 1936
- Penfield, W., and Rasmussen, T.: The Cerebral
Cortex of Man (Lane Medical Lectures,
1947), New York, 1950, The Macmillan
Company.
- Penfield, Wilder, and Shaver, Murton: The In-
cidence of Traumatic Epilepsy and Head-
ache After Head Injury in Civil Practice,
A Research Nerv. & Ment. Dis., Proc.
(1943) 24: 620-634, 1945.
- Putnam, Tracy J., and Cushing, Harvey. Chronic
Subdural Hematoma, Its Pathology, Its Rela-
tion to Pachymeningitis Hemorrhagica and
Its Surgical Treatment, Arch. Surg 11: 329-
393, 1925
- Rand, Carl W., and Reeves, David L.: Dermoid
and Epidermoid Tumors (Cholesteatomas) of
the Central Nervous System Report of
Twenty-Three Cases, Arch. Surg 46: 350-376,
1943.
- Reid, W. L., and Cone, W. V.: The Mechanism
of Fixed Dilatation of the Pupil Resulting
From Ipsilateral Cerebral Compression, J. A.
M. A 112: 2030-2034, 1939
- Rizzoli, Hugo V., McCune, W S., and Sherman,
Irving J.: Surgical Management of Meta-
static Brain Abscess, J Neurosurg 5: 372-
384, 1948
- Rupp, Charles: Metastatic Tumors of the Cen-
tral Nervous System I. Intracerebral Metas-
tases as the Only Evidence of Dissemination
of Visceral Cancer, Arch Neurol. & Psychiat.
59: 635-645, 1948.
- Russell, Dorothy S. Observations on the Pa-
thology of Hydrocephalus, Medical Research
Council Special Report Series No 265.
London, Hm Majesty's Stationary Office, p.
138, 1949
- Russell, Dorothy S., and Donald, Charles: The
Mechanism of Internal Hydrocephalus in
Spina Bifida, Brain 58 203-215, 1935.
- Schmorl, G.: Zur pathologischen Anatomie der
Wirbelsäule, Klin. Wchnschr. 8: 1243-1249,
1929.

- Dandy, W E: Benign Encapsulated Tumors in the Lateral Ventricles of the Brain. Diagnosis and Treatment, Baltimore, 1934, The Williams & Wilkins Company
- Dandy, W E: *Surgery of the Brain*. A Monograph From Volume XII, Lewis' Practice of Surgery, Hagerstown, Md, 1945, W F Prior Company
- Dandy, W E, and Blackfan, K D: An Experimental and Clinical Study of Internal Hydrocephalus, *J A M A* 61: 2216-2217, 1913
- Dandy, W E, and Blackfan, K D: Internal Hydrocephalus, and Experimental, Clinical and Pathological Study, *Am J Dis Child* 8: 406-482, 1914
- Denny-Brown, D, and Russell, W R: Experimental Cerebral Concussion, *Brain* 64: 93-164, 1941
- Dow, Robert, S, Ulett, George, and Raaf, John: Electroencephalographic Studies in Head Injuries, *J Neurosurg* 2: 154-169, 1945
- Elsberg, Charles A: Diagnosis and Treatment of Surgical Diseases of the Spinal Cord and Its Membranes, Philadelphia, 1916, W B Saunders Company
- Elvidge, Arthur, R: The Cerebral Vessels Studied by Angiography, *A Research Nerv & Ment Dis, Proc* (1937) 18: 110-149, 1938
- Elvidge, Arthur R: The Post-Traumatic Convulsive and Allied States, in Brock, Samuel, editor: *Injuries of the Brain and Spinal Cord and Their Coverings*, ed 3, Baltimore, 1919, The Williams & Wilkins Company, chap 11, pp 257-297
- Elvidge, Arthur R, and Baldwin, Matland: Clinical Analysis of Eighty-Eight Cases of Metastatic Carcinoma Involving the Central Nervous System With an Outline of Therapeutic Principles, *J Neurosurg* 6: 495-502, 1949
- Elvidge, Arthur R, and Baxter, Hamilton: Treatment of Multiple Fractures of the Facial Bones With an External Pin Fixation Splint, *McGill M J* 15: 469-475, 1944
- Elvidge, Arthur R, and Jackson, Ira: Subdural Hematoma and Effusion in Infants, *Am J Dis Child* 78: 635-658, 1949
- Elvidge, Arthur, Penfield, Wilder, and Cone, William: The Gliomas of the Central Nervous System, a Study of 210 Verified Cases, *A Research Nerv & Ment Dis, Proc* (1935) 16: 107-181, 1937
- Engeset, Arne: Cerebral Angiography, with Perabrodil (Carotis Angiography), *Acta Radiol, Oslo, Suppl* 56, p 207, 1944
- Engeset, Arne: On Roentgen Examinations in Head Trauma, *Acta Radiol* 27: 481-494, 1946
- Fischer-Brügge, Prof Dr E: Das Klinsanten-syndrom. (Zugleich ein Beitrag über die Entstehung der gleichseitigen Pupillenverweiterung und starre.) *Acta Neurochirurgica, Wien, Springer-Verlag, Band 2, Heft 1: 36-68, 1951*
- Foerster, O.: The Dermatomes in Man, *Brain* 56: 1-39, 1933
- Giblin, N, and Alley, A.: Studies in Skull Growth. Coronal Suture Fixation, *Anat. Rec* 88: 143-153, 1944
- Givré, Alfredo, and Olivecrona, Herbert: Surgical Experiences With Acoustic Tumors, *J Neurosurg* 6: 396-407, 1919
- Grant, W. T, and Cone, W. V.: Graduated Jugular Compression in the Lumbar Manometric Test for Spinal Subarathnoid Block, *Arch Neurol. & Psychiat.* 32: 1191-1201, 1933.
- Green, John R, and Arana, Roman: Cerebral Angiography. A Clinical Evaluation Based on 107 Cases, *Am J Roentgenol.* 59: 617-650, 1948
- Groat, R. A, Windle, W. F., and Magoun, H W: Functional and Structural Changes in the Monkey's Brain During and After Concussion, *J Neurosurg* 2: 26-35, 1945
- Holbourn, A H S: The Mechanics of Trauma With Special Reference to Herniation of Cerebral Tissue, *J. Neurosurg.* 1: 190-200, 1944
- Hamby, Wallace, B: Tumors in the Spinal Canal in Childhood. II Analysis of the Literature of a Subsequent Decade (1933-1942), Report of a Case of Meningitis Due to an Intramedullary Epidermoid Communicating With a Dermal Sinus, *J Neuropath & Exper Neurol* 3: 397-412, 1944
- Haymaker, Webb, and Woodhall, Barnes: Peripheral Nerve Injuries: Principles of Diagnosis, Philadelphia, 1945, W B Saunders Company
- Head, Henry, et al: *Studies in Neurology*, London, 1920, Oxford University Press
- Ingraham, Franc D, et al: Spina Bifida and Cranium Bifidum. Papers reprinted from the New England Journal of Medicine, with the Addition of a Comprehensive Bibliography, Cambridge, Mass, 1944, Harvard University Press
- Ingraham, Franc D, Alexander Eben, Jr, and Matson, Donald, D: Clinical Studies in Craniosynostosis. Analysis of Fifty Cases and Description of a Method of Surgical Treatment, *Surgery* 24: 518-541, 1948
- Ingraham, F. D, and Heyl, K L: Subdural Hematoma in Infancy and Childhood, *J A M A* 112: 198-204, 1939
- Ingraham, Franc D, and Matson, D D: Subdural Hematoma in Infancy, *J Pediat* 24: 1-37, 1944
- Ingraham, Franc D, Matson, Donald, D, and Alexander, Eben, Jr: Experimental Observations in the Treatment of Craniosynostosis, *Surgery* 23: 252-268, 1948
- Jasper, Herbert, Kershman, John, and Elvidge, Arthur: Electroencephalographic Studies of Injury to the Head, *Arch. Neurol & Psychiat* 44: 328-330, 1940
- Jasper, Herbert, Kershman, John, and Elvidge, Arthur: Electroencephalography in Head Injury. Trauma of the Central Nervous System, *A Research Nerv & Ment Dis, Proc* (1943) 24: 388-420, 1945
- King, J E J: Acute Metastatic Brain Abscess, *South. Surgeon* 5: 407-437, 1936
- LeBeau, J: Radical Surgery and Penicillin in Brain Abscess: a Method of Treatment in One Stage With Special Reference to the Cure of Three Thoracogenic Cases, *J. Neurosurg* 3: 359-374, 1946

CHAPTER XI

DISEASES OF THE FACE, MOUTH, AND NECK

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DISEASES OF THE FACE

Facial Injuries

Traumatic lesions of the face are extremely common in civil life due to the hazards of industry, car accidents, and household mishaps. Contusions, especially about the eyes, produce much ecchymosis and swelling of the soft tissues, causing the familiar "black eye." Fractures of the nose or facial bones may accompany facial injuries, and they must always be looked for after blows about the face. Fractures of the nose or facial bones may be diagnosed by inspection and palpation, but x-rays must be taken to confirm the presence and extent of the fractures.

WOUNDS

Open wounds of the face bleed freely, and this free blood supply is helpful in giving rapid healing, and in increasing resistance of the tissues to infection. The facial nerve or the parotid duct may be severed, and this possibility should always be borne in mind. Operative wounds of the face should be made in natural creases, or parallel to Langer's lines, when possible. Sometimes operative wounds may be placed above the hairline or in the eyebrow. Penetrating wounds may produce a large hematoma in the orbit, with proptosis of the eye.

Treatment.—Severe swelling of the face and eyes after contusions may be treated by pressure dressings and cold compresses. After evaluation of the damage, wounds are carefully cleansed, contused and irregular wound edges are excised, and the edges are accurately approximated with the finest pos-

sible nonabsorbable sutures. Suture of muscles and deep tissues to remove tension from the skin will usually allow the skin sutures to be removed by the third or fourth day. Accurate alignment in closure of wounds of the lip, eyelids and eyebrows is especially important. Primary suture of a main trunk of the facial nerve may be possible, but repair of the finer filaments is impractical. A severed parotid duct should be sutured if possible. If this cannot be done, the duct may be transplanted to the mucous surface of the cheek, thus avoiding an external salivary fistula. If neither of these maneuvers can be carried out, simple ligation of the proximal end of the duct may prove satisfactory. Nasal fractures should be molded into shape early. Depressed fractures of the superior maxilla or malar bone should be elevated promptly.

Burns and Scalds

The face, being uncovered, is exposed to burns and scalds, which may be of any degree. These burns, if deeper than first degree, are very disfiguring, and they may leave horrible deformities. Second degree burns heal without contractures, but there are often unsightly scars, with irregularities of pigmentation of the face. Deeper burns usually leave marked facial deformities, often with severe contractures about the mouth, eyelids or neck. Although function of the features of the face may be restored by skin grafts and pedicle flaps, this treatment is time-consuming. In many cases it is impossible to obtain satisfactory cosmetic results, because of differences in color texture of the skin, and hair distribution of

- Sher
- 1894.
- Sherrington, C. S.: *Phil Tr. London, Series B* 190: 45-186, 1898
- Spurling, R. Glen: *Peripheral Nerve Injury—Technical Considerations, J Neurosurg.* 1: 133-148, 1944
- Stewart, Oscar, W.: *The Neurogenic Bladder, Combined Tidal Irrigator and Cystometer, Lancet* 242: 287-289, 1942
- Stookey, Byron, P.: *Surgical and Mechanical Treatment of Peripheral Nerves, Philadelphia, 1922, W B Saunders Company*
- Stookey, Byron, P.: *Compression of the Spinal Cord Due to Ventral Extradural Cervical Chondromas, Arch Neurol & Psychiat* 20: 275-291, 1928.
- Stookey, Byron, P.: *Compression of Spinal Cord and Nerve Roots by Herniation of the Nucleus Pulposus in the Cervical Region, Arch Surg* 40: 417-432, 1940
- Sunderland, Sydney: *A Classification of Peripheral Nerve Injuries Producing Loss of Function, Brain* 74: 491-516, 1951
- Torkildsen, A.: *Ventriculocisternostomy. A Palliative Operation in Different Types of Non-Communicating Hydrocephalus, Oslo, 1947, Johan Grundt Tanum Forlag.*
- Torkildsen, A.: *Carotid Angiography With Special Reference to the Diagnosis of Cerebral Gliomas, Acta psychiat. et Neurol (supp. 55) pp. 3-168, 1949; Arq neuro-psiquiat* 8. L22, 1950 (in English).
- Vincent, Clovis, David, Marcel, and Askénasy, Harden: *Sur une méthode de traitement des abcès subaigus et chroniques des hémisphères Cérébraux, J. de chir.* 49: 1-46, 1937.
- Walker, A. E., Kollros, J. J., and Case, T. J.: *The Physiological Basis of Concussion, J Neurosurg* 1: 103-116, 1944
- Weed, L. H.: *The Development of the Cerebro-Spinal Spaces in Pig and in Man, Contrib Embryol. vol 5, no. 14, Carnegie Institution of Washington, Publication No 225, p 116, 1917.*
- Weed, L. H.: *Cerebrospinal Fluid, Physiol. Rev* 2: 171-203, 1922.
- Williams, D., and Denny-Brown, D.: *Cerebral Electrical Changes in Experimental Concussion, Brain* 64: 223-238, 1911

Infections about the mouth, chin, submental or submaxillary areas may be due to a spread of an alveolar abscess. Such an abscess, after rupture, may persist as a sinus on the face, which heals only after extraction of the offending tooth.

Treatment.—The availability of antibiotics has greatly modified treatment of facial infections, as one wishes to cure the infection and at the same time prevent operative scars. Most acute pyogenic infections can be controlled by the use of hot fomentations and penicillin. Minor incisions to drain softened abscesses may hasten cure, but it will seldom be necessary to perform a mutilating excision of a carbuncle if modern treatment is available. Erysipelas responds spectacularly to penicillin therapy.

Impetigo Contagiosa

This troublesome pustular skin infection is contagious at all ages, but especially in children. The infection can run through a school or a children's ward, attacking many. It first appears as small vesicles on the face or body, or about a wound. These vesicles soon become pustular, the pustules rupture and coalesce with other sorts to form crusted patches. If untreated, the lesions become widespread. The crusts are best removed by hot saline soaks, and the underlying lesions should be treated by applications of penicillin ointment for not more than a five-day period, or with ammoniated mercury ointment. Great care is taken to prevent spread of infection to other areas by avoidance of scratching and handling the sores. Personal contact with others and the common use of towels is avoided, and careful destruction of dressings is indicated. Prolonged use of penicillin may cause sensitization with urticaria and other rashes. Intramuscular antibiotics may be used for short periods.

Chronic infections of the face are caused by syphilis, tuberculosis, actinomycosis, and blastomycosis. A rare spreading acute infection of the face may be due to anthrax.

Benign Tumors of the Face

SEBACEOUS CYSTS

Sebaceous glands are so numerous in the facial skin that it is not surprising that sebaceous cysts are so common in this area. They may appear in any part of the face or scalp, and they are particularly common behind the ears. Sebaceous cysts of the scalp are called wens. Sebaceous cysts are in close contact with the overlying skin, and when uninfected, they have a relatively tough cyst wall. As the cysts enlarge the skin becomes thinned out, and the cysts frequently become infected, forming a small abscess, or a persistent discharging sinus. Rarely, a cyst may open to the skin surface, where the sebaceous secretion becomes dried as a cutaneous horn. Such cutaneous horns may develop malignant changes in the base of the cyst.

Treatment.—Infected cysts should be incised and drained, with excision at a later date. Small clean cysts are to be excised and closed by primary suture. Large cysts of the face are best incised and drained for two or three weeks until they have shrunk to a point where they can be excised with minor scarring. Malignant cutaneous horns should be widely excised; if benign, more conservative excision of the horn and underlying cyst will be sufficient.

DERMOID CYSTS

A dermoid cyst is a rather flat, circular, rubbery tumor, not adherent to the skin, but relatively fixed to the underlying tissue. These cysts are usually found where two ectodermal surfaces fuse during development. In the face they are most frequently found at the root of the nose, or on the forehead at the outer border of the eyebrow. Dermoid cysts may occasionally become infected, forming small abscesses or sinuses from which hair may protrude.

Treatment.—Treatment of these cysts should not be undertaken lightly, as they are attached to the periosteum, or even to the dura mater. The cyst should be excised,

the transplanted tissue. All are improved, though frequently only to a limited degree.

Treatment.—The usual treatment for systemic burn reactions should be instituted if the burns are extensive. The preferred local treatment consists in cleansing of the face, frequent applications of moist normal saline, or the application of a bland ointment. Antibiotics should be used as required. Some surgeons advocate cleansing of the burn, then leaving the surface exposed until a brown eschar forms. If full thickness loss has not occurred, healing will proceed, and the crust will separate in about two weeks.

or other bacteria. The acne may be aggravated by improper cleanliness, cosmetics, squeezing or pressing of the pimples or comedones which often progress to furuncles or carbuncles. Minor infections about the nostrils or upper lip after a head cold may initiate an attack of erysipelas. The terror of facial infections has been reduced since the introduction of the antibiotics, though such infections should still be treated by the physician with great respect. Edema of the face and eyelids is extensive in any major facial infection. A virulent infection in a patient with low resistance may still spread



Fig 118—Carbuncle on forehead with secondary swelling about orbit and eyelids

Infections

The face is prone to all the infections which attack the skin in general, but especially to acne, furuncles, carbuncles, cellulitis, and erysipelas. The thick, exposed facial skin, rich in sebum-producing glands, gives a special character to the infections. Acne, beginning at the age of puberty, is common in both sexes, and the usual site for acne is the face. The essential lesion is the comedo, easily infected with staphylococci

along the course of the facial, angular, and ophthalmic veins to produce a thrombosis of the cavernous sinus, with its marked morbidity and mortality.

The carbuncle, common in the upper lip, begins as a furuncle, but it spreads rapidly to give the characteristic hard edema with multiple necrotic foci. A tender enlargement of the regional glands usually accompanies this infection. An early start in active treatment may prevent secondary abscesses in these glands.

Excision of hemangiomas of the face leaves ugly scars, and complete removal is difficult as the outlines of the tumor are not sharply delimited. The operation is associated with free bleeding. Implantation of radon seeds in the larger and thicker hemangiomas sets up multiple foci of thrombosis which should produce a cure. Injection of sclerosing fluids such as Sodium Morrhuate is less satisfactory. Superficial hemangiomas may be sclerosed by surface radiation with radium or x-ray, or they may be treated by freezing with carbon dioxide snow.



Fig 120—Circloid hemangioma of the face

3. **Circloid Hemangioma** is a rare and more serious form of hemangioma, consisting of a pulsating mass of thickened, dilated blood vessels that have established communication with surrounding normal blood vessels. They are found about the face and scalp most commonly.

This hemangioma is really a form of arteriovenous aneurysm. Excision of the whole lesion with ligation of surrounding vessels may cure the disease, but bleeding is usually very profuse during the operation. Large amounts of blood for transfusion should be available before beginning such an operation.

MOLES

Moles are common about the face and neck. They may be white, hairy, or show varying degrees of pigmentation. Most moles are benign, but occasionally a melanoma may undergo malignant changes. Such melanocarcinomas are among the most lethal tumors, often spreading slowly, but eventually causing death through wide dissemination. There is a popular belief, not limited to the laity, that surgical excision of a mole may lead to malignant changes. When such a train of events occurs, it will be found that the original excised tissue was already a malignant tumor.

Unightly moles, or melanomas subject to irritation should be completely excised. A melanoma which has started to increase in size must be widely excised and the specimen must be submitted to the pathologist for examination, because such a mole may already be malignant. In malignant cases wide excision of the local tumor and of metastatic glands is the only treatment available. Such tumors are extremely resistant to x-ray treatment.

Malignant Tumors of the Face

Cancer of the skin is the most common form of cancer. Mortality statistics do not show this preponderance, because so many of these cases can be cured. The exposed skin surfaces of the face, ears, neck and hands which are open to the sun's rays are the most common sites of skin cancer. Farmers and sailors who have been subject to sun and weather for years very often develop skin cancer. Persons with fair skins more frequently suffer from senile keratoses and skin cancer than those of darker complexion. Skin atrophy, skin tuberculosis, and x-ray dermatitis are precancerous conditions.

BASAL CELL CARCINOMA (RODENT ULCER)

The face is the most common site of basal cell carcinoma, and basal cell carcinoma is the most common malignant tumor of the

followed by suture of the skin. To prevent collection of blood and serum in the wound, a small rubber drain may be left in place for 24 hours. Pressure dressings covering the eyes may be advisable to prevent ecchymosis and swelling of the eyelids. Care should be taken to place the incision in the eyebrow, where possible.



Fig 119—Dermoid cyst of face

RHINOPHYMA

Chronic and recurrent inflammation of the sebaceous glands in the skin of the nose finally produces a large, bulbous, nodular, red swelling of the nose called rhinophyma or "potato nose." It may follow an intractable rosacea. This disease is almost unknown in women. It is really a mass of sebaceous adenomas. Those afflicted with this condition complain of recurrent infections, obstruction to breathing, and the comment and ridicule of acquaintances.

Treatment.—Operation should be performed under local anesthesia to reduce the copious bleeding. The nodular swelling may

be shaved down to normal contour, but some covering should be left over nasal bones and cartilage to aid in regeneration of epithelium. Healing occurs from the base of the sebaceous cysts, and skin grafting may not be required. The operation of shaving away excess tissue may result in marked scar contracture and eversion of the nostrils. The trend at present is to remove all rhinophymatoid tissue and to apply a full thickness skin graft taken from the auricular or clavicular area.

HEMANGIOMA

Hemangiomas, congenital malformations of blood vessels, vary in their clinical appearance. Most of them appear soon after birth. There are three varieties.

1. **Capillary Hemangioma or Port Wine Stain** appears as a red to purple discoloration of the skin, it shows little tendency to increase in size, but it persists through life. When it occurs on the face, there may be a very ugly blemish.

The treatment of capillary hemangiomas of the face is very unsatisfactory. Excision and skin grafting usually leave a deformity little better than the original condition. Disguising of the blemish by pastes and powders is usually preferable to surgical interference. Grenz ray treatment may make the deformity less conspicuous.

2. **Cavernous Hemangioma** is made up of larger blood spaces which usually appear soon after birth, but they may increase rapidly in size, and treatment should be instituted as soon as possible. They may at first grow entirely subcutaneously, showing no discoloration of the skin, but later a bluish raised tumor of the skin follows. Such hemangiomas sometimes ulcerate and become infected. Thrombosis may occur with possible spontaneous cure. Even after cure of those hemangiomas which involve the skin, a white nonpigmented, hairless area persists at the point of the former lesion. Malignant changes very rarely occur.

Leukoplakia frequently appears as a thick, white patch on the vermilion border of the lips, similar to leukoplakia elsewhere. It is difficult to eradicate by irradiation or excision. Cases of leukoplakia should be kept under observation, as hyperkeratosis, papilloma, or cancer may develop. A biopsy must be taken in suspicious cases.

Syphilis

The lip is the most common site of extragenital chancre. This lip lesion is similar in appearance to a chancre elsewhere. There may be great edema of the lip, with enlargement of the regional lymph nodes. A chancre is more often found on the upper lip. Definite diagnosis is made by a search for spirochetes, by a biopsy if necessary, and by the prompt cure of the lip under anti-luetic treatment. Treatment of the syphilis is all that is needed.

Benign tumors of the lips such as warts, polyps, and hemangiomas should be treated by the usual methods. Occasionally such lesions resemble cancer in appearance.

Cancer of the Lips (Epithelioma)

Only those cancers of the lip arising from the vermilion border or the mucocutaneous junction should be included in this category. The cancers which originate in the mucous membrane and spread to the lip, grow more rapidly, metastasize earlier, and have a more serious prognosis. Cancer of the lip is a common tumor, and it comprises about 30 % of oral cancers. At least 95 % of lip cancers appear on the lower lip, less than 5 % occur on the upper lip. Only about 3 % of lip cancers are found in women in most statistical surveys. The majority of lip cancers are found in men over 60 years who have worked for years in the sun. Pipe smoking is not a proved factor in the production of labial cancer.

Cancer of the lip may show itself as a raised, indurated lesion with a small central ulcer; as a deep hard ulcer; or as a raised

warty tumor. This cancer tends to spread along the surface of the lip, rather than to penetrate early into its depth. More than half the lower lip may be attacked by the neoplasm, without deep penetration. Microscopically, the tumor is a typical squamous cell carcinoma.

Metastases from lip cancer occur less frequently than in other forms of oral cancer. Even in the presence of large primary cancers, there may be no glandular metastases. Of lip cancers, 10 % only show glandular spread. Distant metastases are very rare. When metastases do occur, they are found most often in the submaxillary glands.

Exact diagnosis of lip cancers depends on the pathologist's examination of a biopsy specimen. In most cases a biopsy should be done before treatment is initiated.

Treatment.—Except for small cancers of the lip that may be completely excised by a V-shaped removal while doing the biopsy, x-ray therapy is the preferred treatment. The local cancer can be cured in 90 % of cases, with little deformity, by this means. After control of the primary lesion, cervical metastases are best handled by excision, but prophylactic neck dissection to anticipate metastases is not indicated in cancer of the lip. If operation for neck metastases is inadvisable for any reason, the glandular tumor may be retarded by thorough x-ray therapy. In the absence of cervical adenopathy, prophylactic x-ray treatment is not advised.

DISEASES OF THE MOUTH AND TONGUE

The mouth is lined by squamous epithelium, and this epithelium is constantly exposed to minor irritation and injury. The rich blood supply of the mouth is one factor that makes its tissues resistant to the bacteria which are always present. Wounds heal promptly if serious infection is not present.

face These tumors, like epidermoid cancer, tend to occur in the older age groups. They may appear in any part of the face, but most often above the mouth and below the hair line, and about the ears. At the onset the tumor appears as a pearly nodule in the skin, and this nodule in course of time breaks down to form an ulcer with rolled pearly edges. The ulcer enlarges slowly and persistently, and it may finally invade deeply, destroying underlying bone and cartilage, eventually causing hideous deformities. Despite its invasive character, a basal cell cancer rarely metastasizes.

Treatment.—Destruction of senile keratoses by electrocoagulation or excision will prevent the onset of many basal cell cancers. Because of its slow growth, the basal cell cancer is easily controlled in its early stages by adequate x-ray or radium treatment, or by surgical excision or coagulation. The requisite for successful treatment is destruction of all tumor cells. The cure rates of surgery and irradiation are equally satisfactory if properly applied at a reasonably early stage.

EPIDERMOID CANCER

Epidermoid cancers frequently begin in hyperkeratoses of the skin of the cheeks, malar and temporal areas, and ears. They are less common than basal cell cancers on the forehead, eyelids, and nose. The epidermoid or squamous cell cancer usually begins as a warty area, which later ulcerates. It tends to grow rapidly. The edges of the ulcer are indurated, undermined, and infected. These cancers may metastasize to regional glands, though only a small proportion of such tumors of the face metastasize at an early stage.

Treatment.—The primary tumor may be treated by excision or by irradiation. Preference should be given to x-ray therapy if excision will produce large deformities about the eyes, mouth or nose. Tumors that recur after radiation should be treated surgically. Metastatic glands are best treated surgically.

Diseases of the Lips

MACROCHEILIA

Macrocheilia, hypertrophy of the lips, is usually a congenital condition, analogous to macroglossia. It is due to lymphangiectasis, accompanied by a connective tissue overgrowth. The lower lip is more frequently involved than the upper lip. The lip is firm, thickened, enlarged, and everted, causing considerable deformity. It is best treated by wedge excision of the excess tissue.

HERPES LABIALIS

Herpes of the lip generally follows fever, the common cold or exposure to sun. The cold sores begin as numerous small vesicles, which later coalesce to form pustules, followed by a dry crust. The herpes should be completely healed in one to three weeks, unless secondarily infected. The disease is now considered to be due to a virus infection. Herpes and ulcers of the lips which do not heal, under treatment, in a month should be regarded with suspicion.

In the early stages treatment by mild astringents, such as Witch Hazel, may be used. Bland ointments to soften the crusts should be applied later. Local applications of 1% aureomycin in cellulose acetate may be used with benefit.

BENIGN LESIONS OF THE LIPS

Cracks and fissures of the lips may be persistent and annoying, the crack tending to split open on smiling or stretching the lips. Cancer of the lip may begin in one of these fissures. If the crack or ulcer does not heal promptly on treatment by bland ointments, the fissure should be excised, and the specimen should be examined by the pathologist. Chapped lips are associated with cold weather, the use of cosmetics, and the habit of licking the lips. Ointments should be used to keep the lips soft. Cracks about the corners of the mouth may indicate congenital or late syphilis.

GLOSSITIS

Glossitis, or inflammation of the tongue, appears in several forms, and its association with stomatitis and Vincent's angina has already been noted. Acute parenchymatous glossitis or deep inflammation of the tongue arises from penetrating or incised wounds or bite. The onset of inflammation is sudden, the tongue becomes painful and swollen, and it may protrude from the open mouth. Edema may be so severe as to interfere with respiration. Suppuration is not the rule. Treatment includes warm mouth washes and antibiotics. If an abscess forms, it must be incised. In rare cases a tracheotomy is necessary.

LEUKOPLAKIA

Leukoplakia is characterized by proliferation and thickening of the mucous membrane of the tongue or mouth, without evidence of neoplastic changes. The mucous membrane is white, thick and smooth. In later stages it may be warty or fissured. In itself leukoplakia is usually symptomless, but it should always be regarded as potentially dangerous. Cancer develops in patches of leukoplakia much more commonly than in normal mucous membrane. Leukoplakia is seen more often in people over 40 years of age. The etiology is unknown, but chronic irritation, syphilis, poor mouth hygiene, smoking and tobacco chewing, may play a part in its production.

Treatment is unsatisfactory. Oral hygiene should be improved; smoking, intake of spicy foods and alcohol should be curtailed. Syphilis, if present, must be treated. Recurrence of the leukoplakia is to be expected after treatment by excision, cauterization or irradiation. Patients with leukoplakia should be observed regularly so that warts, ulcers and beginning cancers can be treated at the earliest opportunity.

ULCERS OF THE TONGUE

Simple ulcers of the mouth and tongue may be caused by jagged teeth, badly fitting

dentures, other trauma, or from unknown causes. They should be treated by removal of any known cause, by bland mouthwashes, or by painting with dilute solution of silver nitrate. Ulcers that fail to show signs of healing in three weeks should be biopsied. A diet high in vitamins should be prescribed.

SYPHILIS

A primary chancre may appear on the tongue, and it differs little from chancre elsewhere. The regional glands are usually enlarged. Mucous patches of secondary syphilis occur in the mouth. Gumma of the tongue may appear in late syphilis. The gumma of the tongue is usually found near the midline as a firm nodule in its depths, and ulceration may occur later. The blood Wassermann and biopsy will confirm the diagnosis. Specific systemic treatment cures the local lesion.

TUBERCULOSIS

Tuberculosis of the tongue is almost invariably accompanied by other manifestations of the disease in the larynx or lungs. The tuberculous ulcer is generally found on the tip or dorsum. The ulcer is superficial, with undermined edges, a pale necrotic base, and is characteristically painful. Diagnosis may be confirmed by biopsy. Treatment of the general disease will heal the local ulcer, but excision may hasten healing and relieve pain.

ACTINOMYCOSIS

Primary actinomycosis appears, rarely, as a firm nodule in the depths of the tongue. The nodule breaks down to form a necrotic ulcer, or numerous discharging sinuses on the dorsum of the tongue. The cervical glands are not typically enlarged. Carious teeth and pyorrhea are usually present. When the ulcer is excised the fungus may be seen in the stained sections. Excision of the ulcer, extraction of diseased teeth and massive doses of penicillin should cure the disease.

Malformations

Malformations of the mouth include: cleft palate, absence of the tongue, bifid tongue, tongue-tie, macroglossia, fissured or geographic tongue. Malformations of the jaws, and irregularities of the teeth are common and varied. The more severe cases may require surgical treatment, but most cases can be greatly improved by the orthodontist if treatment is begun at a sufficiently early age.

Tongue-tie or ankyloglossia, an abnormal fixation of the tongue, may be congenital or acquired. The congenital variety is usually caused by a short frenum. This may interfere with nursing in the infant, or it may produce speech defects in older children. Simple incision of the frenum relieves the symptoms if carried out early, and this should be done before the child begins to talk.

Injuries

Wounds of the mouth and tongue are frequent, often caused by biting of the tongue. Bleeding may be severe and prolonged. Fractures of the jaws are usually compounded into the mouth and this may lead to secondary infection. Teeth are often broken or completely avulsed during oral injuries.

Larger bleeding vessels should be ligated, and wounds should be sutured. Loosened teeth do not necessarily require extraction. Fractures of the jaws should be immobilized in the best possible position. If sufficient teeth are present, this is best accomplished by wiring the teeth together to splint the fracture until healing is well under way. The use of antibiotics assists in controlling infection.

Burns and scalds of the mouth and tongue, unless very deep, produce only superficial destruction of the mucous membrane and papillae. They are very painful. Deeper burns may lead to the formation of ulcers which heal with a scar. Strong acids or al-

kalis usually cause more destruction in the base of the tongue and the pharynx than in the anterior part of the tongue and mouth.

Inflammations and Infections

Infections in the mouth are very common, with associated spread to the regional lymph glands, or cellulitis of the surrounding soft tissues. Dental abscesses are frequently the primary site of the infection.

STOMATITIS

The term stomatitis is applied to inflammation of the buccal mucosa. The mucous membrane becomes red, swollen, and tender, and there may be marked swelling of the face and lips. Many etiological factors have been suggested, including vitamin deficiency, fevers, local infections from pyorrhea or dental caries. Treatment with heavy metals such as mercury may produce stomatitis. Frequent mouthwashes, antibiotics, and removal of tartar from the teeth aid in the cure.

Aphthous stomatitis or canker appears in children or older persons as pinhead-sized red or yellow spots on the edges of the tongue, or floor of the mouth. They are very painful, and small ulcers may form. The lesions disappear in a few days, but new crops may appear. Cauterization of the cankers with a silver nitrate stick hastens recovery and relieves pain.

VINCENT'S ANGINA (TRENCH MOUTH)

This is an ulcerative form of stomatitis due to an infection with a spirochete and a fusiform bacillus. It involves the oral mucous membrane. Smears should show the characteristic infecting organisms. Treatment includes attention to infection about teeth and gums; mouth washes of dilute potassium permanganate or sodium perborate, and bland diet, which give satisfactory results. Penicillin tablets containing 20,000 units, used every few hours will cure the disease in a few days if oral hygiene is attended to at the same time.

the cyst to the mucous membrane of the floor of the mouth, in effect a marsupialization.

Mixed Cell Tumors similar to those of the parotid gland occur in the mouth, usually on the hard palate. The tumors are well circumscribed and are covered by normal mucous membrane. They grow slowly and painlessly, and they are almost always benign. They can be easily shelled out, and they do not recur if completely removed.

Exostoses of the superior maxilla or palate may protrude into the mouth, especially at the point where the hard palate meets in the midline. The exostosis does not usually require treatment. A true osteoma of the maxilla may be so large as to require excision.

Epulis is one of the commonest tumors of the mouth and is found on the alveolar ridges at the gingival margins of the teeth, often where teeth have been extracted. An epulis usually has a rather narrow attachment, it spreads over the gum surface, and it may hide the crowns of adjacent teeth. It may be a fibroma or it may contain giant cells. The epulis is almost always a benign tumor, which grows slowly and painlessly, and it should not recur if removed completely with the teeth adjacent to the tumor.

Cysts of Jaws

Tumors that contain fluid or semifluid material and have a definite wall or lining membrane are called cysts. Those commonly found in the jaws are:

The **dentoperiosteal cyst** is the type most frequently found and almost always occurs around the root of a tooth following death of the dental pulp. These cysts contain a straw-colored fluid.

Diagnosis of small cysts may only be made by x-ray examination which shows a translucent area with well-defined margins. Large cysts cause bulging of bony contour, and if rupture has occurred, infection with discharge of pus follows. Roentgenograms show large radiolucent shadows which may involve many teeth or invade the antrum.

The nonvital teeth should be removed. The most satisfactory treatment is to raise a mucoperiosteal flap, remove bony wall if present, and extirpate the entire sac. The flap is then sutured back in place, leaving the cavity to fill with blood clot and organize. Another method is to marsupialize the cyst cavity, leaving the lining of the deepest part of the cyst and turning in the mucoperiosteal flap.

In **dentigerous cysts**, the cyst lining is formed from the outer layer of the enamel organ. A significant finding is the absence of a tooth from the dentition and the presence of a painless slowly increasing swelling. The x-rays show a translucent single cavity with sharp margins containing an unerupted tooth. The outer wall of the cyst may become so thin that it will bend like a Ping-pong ball on pressure. The treatment is to raise a mucoperiosteal flap on the buccal surface and remove both the entire capsule and tooth, followed by resuture of the flap.

An **Ameloblastoma** is caused by aberrant growth of the cells of the inner layer of the enamel organ. There is usually a history of swelling for some time and the roentgenogram shows multilocular cystic cavities. Some spaces are cystic while others are filled with solid cellular growth. Sometimes it is impossible to distinguish between giant cell tumor and osteitis fibrosa cystica. The treatment of small growths is to remove the abnormal epithelium by enucleation and curettage searching out and removing all cysts. When the tumor is large, with perforation of the bone and extension into soft tissues, the condition should be treated by resection of the involved area. In the mandible when resection is necessary the fragments may be fixed to prevent displacement, and at the same time, or a few months later, a bone graft may be inserted.

Malignant Tumors of the Mouth and Tongue

Cancer within the mouth is common, and it may involve the tongue, cheek, gum mar-

Inflammations of the Teeth, Gums, and Jaws

PYORRHEA ALVEOLARIS

The deposit of decayed food, and tartar about the teeth at their junction with the gums, allows infection to occur under the gum edges. The gums become red, pus escapes, gums recede, and from this source infection may spread to the floor of the mouth or neck. Treatment consists in cleaning tartar from the teeth and oral hygiene.

ALVEOLAR ABSCESS

As a result of tooth decay, bacteria may reach the root canal, causing an abscess at the root of a tooth. The abscess may be symptomless, or there may be tenderness on pressure on the tooth. Such an abscess may lie dormant, or it may break through the thin external plate of the alveolus to produce a chronic sinus. Extraction of the tooth usually cures the root abscess.

An acute alveolar abscess may cause severe pain with swelling of the face or neck, redness of the gums, and tenderness on pressure over the affected tooth. The abscess may rupture into the mouth, or it may cause an inflammation in the face, floor of the mouth, or in the neck. Osteomyelitis of the jaw frequently results if treatment is delayed. The use of antibiotics and tooth extraction will bring about a cure.

Extension of the acute inflammation of the mouth to the neck, as a cellulitis or a cervical adenitis, is to be expected. Abscess formation is common. If the abscess breaks into the submaxillary and submental spaces, a Ludwig's angina may develop with protrusion of the tongue and interference with respiration.

Tumors of the Mouth and Tongue

A BENIGN TUMORS OF THE TONGUE

Dermoid Cysts occur in the tongue, arising from the embryonic rests of the branchial clefts. These cysts are found in the midline of the anterior part of the tongue, or appear in the lateral portions of its base. Occa-

sionally the dermoid persists only as a sinus at the base of the tongue, and this sinus is continuous with a dermoid cyst or sinus in the lateral part of the neck. The cyst or sinus is treated by excision of the whole cyst and sinus.

Other Benign Tongue Tumors.—Rarely, innocent tumors such as lipomas, fibromas and teratomas, containing bone or cartilage, may appear in the tongue. Hemangiomas of the capillary or cavernous types are more common, the cavernous hemangioma sometimes showing as a pedunculated bluish mass which bleeds easily and freely.

Papillomas and warts are quite common on the tongue, and some of these papillomas become large enough to cover a large area of the tongue, suggesting the possibility that cancer has already developed.

All these tumors should be treated by excision, and in all cases the tissue must be examined by a pathologist. An inflammation or hypertrophy of a lingual tonsil may be mistaken for a cancer. This tonsillar tissue is situated on the lateral border of the tongue at its junction with the anterior pillar of the soft palate.

BENIGN TUMORS OF THE MOUTH

Mucous Cysts are small retention cysts, containing thick mucus, which form in the mucous glands of the buccal mucosa. Complete excision cures the cysts.

Ranula is a cystic swelling situated in the floor of the mouth to one side of the midline. Its origin is not clear, but it appears usually to be a cystic dilatation of the sublingual salivary gland. The cyst is usually unilateral, may reach a large size, appears in childhood, grows slowly, and may extend to cause swelling in the submaxillary triangle.

Treatment by complete excision is technically difficult because of the thin, delicate cyst wall, and the large size of the cyst. Suture of the defect after incomplete removal of the ranula will cause a recurrence. Most of the cysts may be cured by unroofing the cyst, and then suturing the cut edges of



Plate II.—Cancer of the Lip.



Plate III.—Leukoplakia of the Tongue.



Plate IV.—Cancer of the Tongue.

gins, floor of the mouth, palate or tonsils. Cancers in these different areas of the mouth vary somewhat in rapidity of growth, malignancy, and tendency to form metastases. In all cases the disease is so malignant and dangerous that the need for early diagnosis and prompt, adequate treatment cannot be overemphasized.

Cancer of the mouth accounts for about 4 % of all cancer in man, and the male is attacked four or five times as commonly as the female. The neoplasm, usually occurring after forty years of age, is most common between the ages of fifty and seventy years.

Although the etiology of cancer is unknown, intraoral cancer is generally found in association with poor mouth hygiene, rough carious teeth, badly fitting dentures, leukoplakia, and syphilis. There is often a history of excessive smoking or of tobacco chewing. Chronic avitaminosis, with glossitis and stomatitis may also be present.

About 90 % of mouth cancers are of the epidermoid type, arising from the squamous epithelium which lines the mouth and covers the tongue. Adenocarcinoma may arise from mucous or small salivary glands, and such cancers are most frequently found in the palate. Melanocarcinomas infrequently appear in the mouth, usually on the palate. Lymphoepitheliomas may originate about the base of the tongue, tonsils, and oropharynx. Most of these tumors tend to metastasize to regional lymph nodes.

CANCER OF THE TONGUE

The mobile anterior two-thirds of the tongue extends from the tip to the vallate papillae. The base of the tongue extends backward from this point to the hyoid bone. The squamous epithelium which covers the tongue is firmly adherent to the underlying muscle. The lymphatics of the anterior part of the tongue drain to the submental and submaxillary triangles, and to the internal jugular chain of glands. The lymphatics of the base of the tongue, in common with

the oropharynx, drain to the digastric glands and to the internal jugular chain. There are lymphatic communications across the midline of the tongue, especially at the tip and base of the organ, so that cancers of the tip, dorsum, or base of the tongue frequently metastasize to both sides of the neck. However, when the growth is confined to the lateral margin of the tongue, unilateral spread only is to be expected.

Cancer of the tongue begins as an indurated area or a fissure in a patch of leukoplakia, as a papilloma, or as a small ulcer. The earliest lesions look so innocent that cancer is often not suspected. Central necrosis and ulceration of the cancer occur early. Severe pain is not usually a symptom in a small tongue cancer, and this fact is responsible for the delay in diagnosis of cancers of the base of the tongue. The cancer begins most often on the lateral margins or under surface of the anterior part of the tongue. The primary lesion, if untreated or uncontrolled, does not long remain superficial, but spreads as a deep burrowing indurated ulcer. In the later stages severe pain, infection, tongue fixation, salivation, cervical metastases, and terminal hemorrhage present a horrible picture.

Examination of the tongue should include palpation, as well as inspection, as the area of induration may be more extensive than might appear from inspection alone. Palpation is especially useful in examination of tumors of the base of the tongue.

Cervical metastases appear early in tongue cancer, and they are present in about 40 % of patients when first examined. Cervical metastases can be expected at some stage in 80 % of cases under observation or treatment, if the primary lesion is not cured, and metastases are frequently bilateral. Distant metastases are considered rather uncommon, but autopsies frequently show them.

The appearance of the lesion, its progressive growth, the age of the patient, leukoplakia, and enlarged cervical glands may make positive diagnosis of cancer easy.

Vincent's angina, traumatic ulcer, papilloma, tuberculosis, or gumma must be considered in the differential diagnosis. Biopsy, which should be done early, is necessary to settle the diagnosis. A positive Wassermann reaction does not eliminate the probability of cancer, and indeed, mouth cancer is much more common in syphilitics than in the general group of the population. All suspicious tongue lesions should be regarded as cancer until proved innocent by biopsy.

Treatment.—Much may be accomplished by prophylaxis to prevent oral cancer. Removal of small areas of leukoplakia, dental care, and the treatment of syphilis are indicated.

When tongue cancer has begun, treatment includes attack on the local lesion and on the metastases. In the advanced cases palliative treatment only is advisable.

Before the discovery of radium and x-rays cancer of the tongue was treated by radical excision of the tongue, with extensive dissection of the cervical glands. The operative mortality was so great, and the recurrence rate was so discouraging, despite mutilating operations, that surgeons willingly referred their cases of tongue cancer to radiotherapists, when these methods were perfected. Except in selected early cases, however, the five-year survival rate after treatment by radiotherapy, in the best hands, is no better than 30 %. Improvements in anesthesia, blood transfusion, and antibiotics have lessened the risk of operation, and the trend is again toward surgical treatment, or a combination of methods, in the hope of improving the survival rate.

Small cancers of the lateral borders or tip of the tongue should be treated by excision of the tumor, with implantation of radium in the wound. This treatment may be supplemented by external x-ray therapy. Cancer of the base of the tongue is still best treated by radiation therapy alone. Hemiglossectomy or total excision of the tongue is advisable in cases with extensive leuko-

plakia, in cases that fail to respond to radiation, or in which the tumor recurs after thorough irradiation.

When the primary tumor in the tongue appears to be controlled, metastatic cervical glands should, if possible, be removed by block dissection. Bilateral excision of glands may be necessary. Failures of treatment of tongue cancer are more often due to the failure to eradicate the metastases. X-ray therapy is very useful in controlling inoperable metastases or recurrences after operation.

It is too soon to be sure that this return to surgical treatment of tongue cancer will improve the survival rate. It must be emphasized that proper surgical treatment of cancer demands complete destruction of the primary growth, and removal of the lymphatics and glands draining the tumor area. Incomplete operations, in most cases, serve only to hasten spread of the disease. Electrocoagulation of fixed mouth tumors, that appear too large for surgical excision, may give surprisingly good results, and this form of treatment has much to recommend it.

CANCER OF THE FLOOR OF THE MOUTH

Most of what has been said about cancer of the tongue applies equally to cancer of the floor of the mouth. The lymphatics of the floor of the mouth drain to the submental and submaxillary triangles, and thence to the internal jugular chain. The tumor appears as a superficial ulcer, or as a crevice with the main mass of the neoplasm growing under the mucosa. The cancer almost always begins on one side; but it may soon cross the midline, spread to the tongue, or become attached to the mandible.

The early lesion is an indurated ulcer in the floor of the mouth. The submaxillary glands are invaded in about a quarter of the cases on first examination. A biopsy, which may include the whole of a small growth, will decide the diagnosis. Inflammation of a submaxillary gland, associated with obstruction of the duct by mucous plugs or calculus, may cause some confusion in diag-

CANCER OF THE MAXILLARY SINUS

The lymphatics of the antrum communicate with those of the nasal fossa, and they end in the retropharyngeal, submaxillary and the jugular lymph glands. Most cancers of the antrum begin in the floor of the sinus near the tooth roots. As it grows the tumor expands the anterolateral wall and the floor of the sinus, giving swelling in the cheek, palate and upper gum, with loosening of the bicusps and anterior molars. This cancer is usually a well-differentiated, squamous carcinoma which grows rather slowly and does not have early metastases. The early symptoms may be toothache or pain in the cheek, with loosening of teeth later. An early diagnosis is seldom made, as the symptoms are thought to be caused by dental caries or sinusitis. Diagnosis is made by examination of the nose, palpation of the palate, gums, and cheek, x-ray of the sinuses, and needle puncture biopsy of the antrum.

Treatment.—If diagnosis can be made before the cancer has broken through the walls of the sinus to the soft tissues, the cure of this lesion is hopeful. At this stage, radical excision of the superior maxilla offers a good chance of complete cure. At a later stage, opening of the sinus, with coagulation of the tumor, and external x-ray therapy, may give five-year cures in 30% of cases.

NECK

Congenital Anomalies

In the embryo there are five branchial arches with four branchial grooves or clefts between them. The arches develop to form the upper and lower jaws, parts of the hyoid bone, the styloid process, part of the stapes, and most of the thyroid and cricoid cartilages. The branchial clefts and furrows form the external auditory meatus, the Eustachian tube and tympanic cavity, the thymus and parathyroids. The major parts of the branchial clefts normally disappear, but they may persist in whole or in part.

BRANCHIAL CYST

The most common remnant of the branchial clefts may form a branchial cyst. A branchial cyst usually appears in early adult life as a smooth, movable, soft, painless mass in the lateral part of the neck near the angle of the mandible under, or anterior to, the sternomastoid muscle. Such cysts are usually lined by squamous epithelium, and contain creamy fluid. Smears of the fluid as a rule contain cholesterol crystals. A few deeper cysts may be lined by columnar epithelium and these may contain mucus. Fluctuation can be felt in some larger cysts



Fig. 121.—Infected branchial cyst with sinus formation.

These cysts should be excised through a transverse cervical incision. During removal of some larger cysts care must be taken not to cut the cervical sympathetic chain, as the deep part of the cyst may be closely related to it.

BRANCHIAL FISTULA

Persistence of a branchial cleft may form a fistula in one or both sides of the neck.

nosis, but there is typically no ulcer in such cases. Benign or malignant salivary gland tumors occur rarely in the floor of the mouth, but they do not usually ulcerate.

Treatment.—Local excision of the primary growth is generally followed by recurrence. If radiation therapy is to be used, extraction of all teeth in the area should be done before beginning treatment. Intraoral x-ray therapy is difficult as the growth is inaccessible. Small cancers may be treated by radium or radon implants in the mouth, and external x-ray treatment. In larger tumors, involving bone, radical excision of the primary tumor with the mandible and glands is to be preferred. Some necrosis of the mandible sometimes follows radiation therapy. In the presence of enlarged cervical glands, neck dissection is indicated. The prognosis in cancer of the floor of the mouth is somewhat better than in cancer of the tongue.

CANCER OF THE BUCCAL MUCOSA

The lymphatics of the mucosa of the cheek follow the facial vein to the submaxillary triangle and the cervical lymph nodes. Cancer of the buccal mucosa is less common than cancer of the tongue or cancer of the floor of the mouth. Buccal cancer generally begins in an area of leukoplakia, and it may appear as a warty, polypoid, or ulcerating tumor. The latter form is the most malignant type. Metastases occur as in other oral cancers.

Early warty types may be successfully excised. Radium or radon implants in the cheek should be supplemented by x-ray therapy in larger growths. More extensive tumors require wide excision with dissection of cervical glands.

CANCER OF THE PALATE

The lymphatics of the palate drain to the deep glands of the neck, but spread to the submaxillary or retropharyngeal glands is possible. A lymphatic spread across the midline to the glands of the opposite side of the neck is frequent. Benign tumors of the palate, of mucous and salivary gland type,

are more common than carcinoma, which is relatively rare. The cancers tend to cover a large area of the palate, with multiple superficial ulcerations, and slow growth. Metastases appear late, but they are frequently bilateral.

Treatment is unsatisfactory. Complete excision is difficult, and the cancer is rather radioresistant. However, intraoral x-ray therapy should be tried. If this is unsuccessful, excision or electrocoagulation should be performed. Either of these latter forms of treatment usually leaves a defect in the hard and soft palate that is hard to close with dentures or by surgery. When the primary lesion is under control, metastatic glands should be excised.

CANCER OF THE TONSIL

Cancer of the tonsil, next to cancer of the laryngopharynx, is the common malignant tumor of the upper air passages, and it comprises about 2 % of all cancers. Malignant tumors of the tonsil may be carcinomas, lymphoepitheliomas or lymphosarcomas. The epidermoid cancer is the commonest form, and it appears as an ulcerating tumor of the tonsil, spreading to the soft palate and the pillars of the tonsil. This cancer is rather undifferentiated, radiosensitive, and it gives early glandular metastases.

The *lymphoepithelioma* is usually a smooth tumor, showing little ulceration or spread to the palate. Glandular metastases appear early, and there may also be extension to the lungs, mediastinum, liver, and bones. This tumor is extremely radiosensitive.

Lymphosarcoma does not ulcerate until late, but it may reach a large size in the tonsil. Enlarged cervical glands accompany the tonsillar hypertrophy, and the disease may give a general lymphadenopathy as in lymphosarcoma elsewhere. Here, too, the disease is very radiosensitive.

Treatment.—Surgical treatment is usually unsuccessful. Both the primary lesions and the metastases are so sensitive that x-ray treatment is the best form of treatment.

give rise to a spreading cellulitis called Ludwig's angina. There is a firm swelling in the submaxillary and submental regions, with edema of the mouth, and usually with protrusion of the tongue. The infection may break from the submaxillary triangle and spread down the neck under the deep fascia. The inflammation may progress to such an extent as to cause edema of the glottis and death from toxemia and asphyxia



Fig 122—Ludwig's angina which has been incised. (Transverse incision gives less unsightly scar)

An incision in the submaxillary and submental areas will drain the first pocket of pus, but further incisions may be necessary if the cellulitis has progressed down the neck. Antibiotics must be used. In rare advanced cases tracheotomy may be indicated to relieve embarrassed respiration

BOILS AND CARBUNCLES

Boils and carbuncles of the back of the neck are extremely common. Friction of collars, growth of hair, and the thickness of the skin make this area subject to infec-

tion. Here it is hard for infection to burrow to the surface; the furuncles are liable to spread as carbuncles. Boils and carbuncles here should be treated as in other locations.

RETROPHARYNGEAL ABSCESS

Pyogenic or tuberculous infection in retropharyngeal glands, most often in children, may break down to form a retropharyngeal abscess. A tuberculous abscess in this site may also be secondary to cervical Pott's disease, with caries of the bone. The symptoms to be expected are fever, swelling in the pharynx, difficulty in breathing, and more or less toxic reaction. Rupture of such an abscess may cause aspiration and asphyxia. The abscess tends to spread behind the prevertebral fascia, and it may point on either side of the neck behind the sternomastoid muscle. An x-ray should be taken to make sure there is no disease of the cervical vertebrae. In treating such cases the aim is to get rid of the obstruction to respiration by evacuating the abscess, without allowing aspiration of pus into the air passages. In the absence of spinal tuberculosis, the abscess should be opened through the mouth. The patient's head should be lowered, suction and a tracheotomy set should be ready in case of need. With these preparations, an incision should be made directly into the pus pocket. The incision may often be made without anesthetic. In the presence of cervical bone disease, the abscess should be aspirated through the side of the neck, or the pus may be evacuated by an incision behind the sternomastoid muscle, with suture of the wound

Slowly enlarging swellings are frequent in the neck, and it is often difficult to decide whether the lesions are due to some chronic inflammation, or to local or metastatic neoplasm. Each case will eventually progress to a point where the general symptoms and local findings will make the diagnosis clear. In the early stages the glandular enlargement due to cervical adenitis, tuberculosis, Hodgkin's disease, lymphosarcoma, leukemia,

The external opening of the fistula is usually found near the insertion of the sternomastoid muscle, above the clavicle. If an internal opening is present, it is found near the tonsillar fossa, but the tract is usually incomplete. Periodic opening and closing of the fistula may lead to recurrent abscesses, and at most times there is some discharge from the external fistula. Injection of Lipiodol into the fistula will show the length of the internal tract. A complete fistula passes between the external and internal carotid arteries and deep to the lateral horn of the hyoid bone. The fistula must be completely excised through one or more transverse incisions in the lateral part of the neck.

CYSTIC HYGROMA

Early in infancy a soft swelling may be noted in the lateral part of the neck, generally in the supraclavicular triangle, but it may extend from the clavicle to the mastoid. The cystic hygroma becomes firmer and more apparent when the child cries or strains. The larger cysts may be translucent. These hygromas may be multiple or solitary cysts, lined by endothelium, and containing lymph. Recurrent inflammation may occur in a hygroma; the cysts may rupture and disappear spontaneously. On account of the thin wall excision is difficult. Operative treatment should be postponed, as regression may take place or the cysts may rupture. As the endothelial lining is relatively radiosensitive, x-ray therapy often helps regression. If the hygroma persists over two years, excision must be undertaken.

Injuries

Wounds of the neck are common and conform to the usual classification. The carotid vessels are protected by the sternomastoid muscles, but deep puncture wounds may easily be fatal due to hemorrhage. Deep wounds may reach many important structures: nerves, blood vessels, and air passages. Packing the wound with gauze may control bleeding until the wound can be well

exposed in the operating room. Cut throat is most often due to an attempt at suicide. The air passages are more likely to be damaged than are the great vessels or nerves. Bleeding, infection, and pneumonia are common complications. The patient may have subcutaneous emphysema. Arteriovenous fistulas can be formed by penetrating wounds of the neck.

Treatment.—Bleeding must be controlled, lacerations of the larynx, pharynx, or trachea should be sutured. A temporary tracheotomy may be necessary. The skin should be sutured in the usual way. If the wound is grossly contaminated, or if there is poor hemostasis, drainage should be instituted. It is wise to use antibiotics freely to prevent wound infection and also to lessen the danger of respiratory complication.

Infections

Acute infections in the neck are very common. They are usually secondary to infections beginning in the scalp, face, mouth or throat. Lymph nodes may become enlarged and tender, and the glands may break down to form abscesses. It is difficult to decide when an abscess has formed, and there is no advantage in cutting into a gland that is still solid. The infection is often so deep that there is no fluctuation, only a solid tender mass. By the time there is redness of the skin an abscess has usually formed. Early treatment with antibiotics will often prevent purulent degeneration of glands, especially if the local abscess in the area is controlled by incision of furuncles, or extraction of the abscessed tooth. Cervical abscesses should be incised as soon as they are evident.

If infection in a lymph node breaks through the gland capsule, it may spread as a cellulitis, contained by the firm fascial planes in the neck. Only after a large abscess has formed does the pus break through to the subcutaneous tissues. Infection reaching the submaxillary triangle from the mouth, often from an alveolar abscess, may

to give sinuses, the fresh pus from a newly opened abscess often showing sulphur granules. If untreated, the disease continues to burrow and produce new thick-walled abscesses and sinuses. Gradually the patient becomes thin and weak, with spread of the disease to other parts such as the liver, lungs and brain. The treatment may include wide local excision of damaged tissue, and massive doses of penicillin and iodides.

Tumors

In the lateral part of the neck there may be benign tumors. Lipomas, sebaceous cysts, dermoid cysts and fibromas in the neck have the same diagnostic signs as in other locations and they should be treated by excision. Other benign lateral tumors are branchial cysts, aberrant thyroid tissue, neurofibromas and carotid body tumors.



Fig 124.—Lipoma of the neck.

CAROTID BODY TUMORS

The carotid bodies, one on either side of the neck, lie close to the bifurcation of the common carotid artery, and the normal gland measures about one half a centimeter in diameter. Tumors may be chromaffinomas and they may be related to the sympathetic

nervous system. There is no agreement as to the percentage of malignancy in these tumors, but probably about 15 % are malignant. The tumor is generally smooth and elastic and it usually lies high in the neck under the sternomastoid muscle. The tumors are occasionally bilateral. A bruit



Fig. 125.—Dermoid cyst of submental region which has been growing slowly for 20 years, causing elevation of tongue and interference with eating.

may sometimes be heard, and there is sometimes a Horner's syndrome. If possible the tumor should be excised, without ligation of carotid vessels; but the carotid vessels may be completely surrounded by the tumor. In older patients ligation of the common carotid artery is liable to produce a hemiplegia, and it is better to leave a benign tumor in place than to cause this paralysis. X-ray therapy may prevent progress of the growth.

HODGKIN'S DISEASE

The first symptom of this disease is usually a painless swelling in the lateral part of the neck, and the mass may at first be solitary. There is a smooth, elastic tumor, less firm than one finds in metastatic can-

and metastatic cancer may have no local signs that are clearly diagnostic. Even after complete physical, x-ray, blood, and laboratory examinations the diagnosis may still be in doubt. An early diagnosis being urgent, biopsy will often be necessary, so that the pathologist's examination of the tissue will allow early rational treatment of the disease. Even the biopsy may sometimes leave the question unsettled. Either the disease has not involved the excised tissue, or it has not yet progressed to a stage when the tissue changes are diagnostic.

Tuberculosis

The most common chronic inflammation of the lymph nodes of the neck is due to tuberculosis, and the neck is the most frequent site of glandular tuberculosis. Although supervision of milk supply and more



Fig 123—Recurrent tuberculous abscess of the neck

rigid control of human tuberculosis have greatly reduced the incidence of glandular tuberculosis, the disease is still not rare. Early in the disease, one, or several, movable, discrete, rubbery glands may be palpated in any of the cervical triangles. In some cases,

chains of matted glands develop which seem to have no tendency to undergo caseation. In most cases, as the glands become progressively larger, they tend to caseate and become fixed to the surrounding tissues and to the skin. Later there may be secondary infection, with redness of the skin and spontaneous rupture of the abscess. Following rupture a sinus may persist. The lesions are usually painless until secondary infection appears, and fever and loss of weight may be entirely absent. While glandular tuberculosis is generally considered to be a disease of youth, this disease is not infrequently found in those of advanced years, especially in women. X-rays may show calcification in the glands.

Treatment.—The treatment of glandular tuberculosis has been changing during the past years, and further changes are likely to occur. It is probable that local enlargement of a gland in the neck is only one manifestation of tuberculosis that exists elsewhere in the body. Wide excision of cervical glands treats only a symptom, and does little to control the underlying cause. In the stage of gland proliferation, and before caseation, x-ray therapy will cause regression of the inflammation. In the stage of caseation, and before secondary infection, local excision of degenerated glands is indicated to prevent sinus formation and to promote rapid healing. After secondary infection the abscess should be opened and the gland remnant should be curetted out. General treatment of the patient with rest, sun, and good food should not be neglected. Streptomycin has been a recent aid in the treatment of gland tuberculosis, and it is probable that more efficient drugs or antibiotics will be available in the near future.

ACTINOMYCOSIS

The majority of cases of human actinomycosis are found in the face and neck. The primary focus of entry is usually in the mouth, around the teeth, and the first sign of neck swelling may follow a tooth extraction. Abscesses soon form and break down

or uterus may be the presence of a firm gland above the left clavicle. A metastatic gland in either supraclavicular area may be an indication of the inoperability of a carcinoma of the bronchus or esophagus. An enlarged cervical gland may be the first obvious spread from a cancer of the testis. Many a patient dates the onset of an intraoral cancer from the time he first noticed a swelling in his neck. Metastases from a tonsil appear under the upper part of the sternomastoid muscle; those of the lip, floor of the mouth, and anterior tongue are found in the submental and submaxillary triangle; while those from the base of the tongue go directly to the internal jugular chain.

Secondary cancer in the cervical lymph nodes may produce a solitary, firm, movable mass, painless in the early stages. The original mass may become quite large before other glands are palpable, and this tumor may become softened in the center, suggesting the possibility that the lesion is inflammatory. Usually a chain of firm nodules soon appears, and before long the glands will be matted together, and fixed to the skin and surrounding tissues. Later the cancer may grow into the skin forming an ulcer, with secondary infection and foul discharge. As the tumor grows and becomes fixed, the patient begins to complain of pain, cancers in the upper cervical areas often causing agonizing pain referred to the ear. Enlarged cervical glands, in the presence of oral cancer, are not necessarily metastases, but may be due to secondary infection which can subside after destruction of the primary tumor.

Treatment should be undertaken before the glands become fixed. If no primary tumor can be discovered, or if there is no history of a pre-existing growth, a biopsy may be necessary. Metastatic lymphoepitheliomas in the neck are radiosensitive, and these rarer tumors should be given x-ray therapy. The majority of neck metastases are secondary to squamous cell cancers of intraoral origin, and they are relatively radioresistant. It is wise to excise these

metastases radically before giving radiation, as a radioresistant cancer may become fixed and inoperable during the time that must elapse while the x-ray treatment is being given, and while the reaction is subsiding to a point where surgical attack on the enlarged glands is possible. However, it is true that, in some cases, inoperable, fixed glands undergo such regression after a course of radiation that surgical excision becomes feasible.

The type of operation used depends on the location of the primary tumor, and on the extent of the cervical metastases. For cancers of the lower lip, tip of the tongue, and floor of the mouth, it may be sufficient to do a suprahyoid dissection of the glands in the submental and submaxillary triangles. Cancers of the posterior two-thirds of the tongue, the tonsils, palate and sinuses will metastasize to the jugular chain. An adequate block dissection in such cases must be patterned after the method of Crile, in which the sternomastoid muscle, the internal jugular vein, and the carotid sheath are removed with all glands and fat in the anterior and posterior triangles of the neck. Postoperative radiation may add to the efficiency of this operation.

Boeck's Sarcoid

Boeck's sarcoid is a chronic indolent benign infectious disease of unknown cause which may involve the lymph glands, skin, salivary glands, lungs, or other organs. The systemic symptoms are usually not severe in the early stages and they may be so mild that only skin eruption on the face or enlarged glands may be noted by the patient. When cervical glands are enlarged, they are rarely more than one inch in diameter. The glands are discrete and elastic, they never caseate or suppurate, but they are involved in a granulomatous inflammation. Preauricular and postauricular glands are frequently enlarged. There may be a hypochromic microcytic anemia. When general lymphadenopathy and splenomegaly are present, the disease may be confused

cer One can expect a progressive enlargement of the tumor, and other masses usually appear. The disease is more frequent in males than in females, the course runs from months to many years, but it is usually fatal in from two to four years. In the final stages there are typically fever, loss of weight, and weakness. The spleen may be enlarged, and x-rays of the chest may show a widening of the mediastinum. The blood picture is variable, but anemia and leukopenia may be expected. At first it is difficult to distinguish Hodgkin's disease from tuberculosis, lymphosarcoma, or cancer. Suppuration does not appear in Hodgkin's disease. Positive diagnosis is made after biopsy of the mass. In a few cases there is no recurrence after excision of the primary tumor. However, the main lesion should be excised, and the operation should be followed up by x-ray therapy, as the disease is very radiosensitive in the early stages. Late cases may be benefited by treatment with nitrogen mustard.

LYMPHOSARCOMA

Lymphosarcoma is also a progressive and fatal disease which may begin with the enlargement of a solitary lymph node in the neck. This solitary tumor may persist for some time, but there is usually a fusion with other nodules to form a matted chain of glands, without suppuration. The reticulum cell sarcoma is probably a variety of lymphosarcoma, but there is no clinical difference between the various forms. The rapidity of progress varies, but this disease is usually more fatal than Hodgkin's disease. A biopsy will settle the diagnosis. Lymphosarcoma is very radiosensitive when it attacks the glands, and x-ray therapy is the best treatment. Usually the tumor recurs rapidly in the original or other areas.

Lymphatic Leukemia often causes the appearance of enlarged, discrete, nonsuppurating glands in the neck. Except in the aleukemic cases, examination of the blood will solve the diagnosis, and in the aleukemic cases a biopsy will usually show the picture of lymphoid infiltration of the nodes.

Carcinoma

The vast majority of carcinomas of the cervical lymph nodes are due to metastases. A very small proportion may be due to primary cancers originating in remnants of the branchial clefts, but a careful search of the scalp, ears, tonsils, pharynx, sinuses and Eustachian tubes may reveal a small primary cancer, at first unsuspected. Careful observation of the patient for years, after excision of a cervical cancer, may finally demonstrate the primary origin. The rare cases of branchial carcinoma usually appear first as a firm tumor under the upper part of the sternomastoid muscle, the mass enlarges and becomes fixed, and spreads as do the metastatic cancers. The mass should be excised as widely as possible, and if the excision does not seem completely satisfactory, it should be followed by a course of x-ray therapy.

SECONDARY CARCINOMA

Cases of Hodgkin's disease, lymphosarcoma, and reticulum cell sarcoma offer an interesting diagnostic problem to the surgeon, but his services are usually dispensed with after the biopsy has been taken and the diagnosis has been established. The radiotherapist gives the patient palliative treatment, but the termination is usually fatal, and the course of the disease is downward over a period which may be long or short. On the other hand, there is some hope for the patient who develops metastatic cancer, secondary to a primary lesion of the skin, lip, mouth, tongue, tonsils, sinuses or thyroid gland. Careful observation of patients after treatment of cancers in these locations may demonstrate metastatic lesions in the cervical glands while they are still small, movable and operable. Surgical excision of the metastases offers the best chance of a permanent cure.

The location of the cervical metastases depends on the site of the primary tumor. The secondaries of cancer of the lung, breast and abdominal organs may appear in the supraclavicular triangle. The first sign of recurrence of a cancer of the stomach, rectum

be removed by an incision in the floor of the mouth. If the calculus is in the submaxillary gland, it is better to excise the gland through an incision in the submaxillary triangle.



Fig 126—X-ray and photograph of salivary calculi.

Tumors of the Salivary Glands

The most common tumor of the parotid gland is the mixed tumor. About 90 % of mixed tumors appear in the parotid gland. These tumors probably arise from epithelium of the salivary gland. Benign tumors may gradually take on malignant changes.



Fig 127.—Mixed tumor of parotid gland.

Salivary gland type tumors may be benign or malignant. They are usually found in the salivary glands, but they may appear in the base of the tongue, the hard or soft palate, the buccal mucosa, floor of the mouth, trachea, lip or bronchus. Salivary gland tumors are found about 12 times more often in the parotid than in other sites. About 20 % of salivary tumors are malignant. About 1 % of all tumors appear in the salivary glands.

The usual symptom of a mixed tumor of the parotid gland is a painless swelling. There is no fixation to the skin and little fixation to the surrounding gland. The clinical impression of mobility of the tumor is deceptive, as the firm subcutaneous bands and the thin capsule of the tumor make removal of the tumor difficult. Pain and facial paralysis are rare in benign tumors. These tumors grow slowly, and most tumors have been present for years before the patient seeks medical help. Mixed cell tumors may undergo degeneration and softening.

Treatment.—The external carotid artery, the posterior facial vein, and the facial nerve are closely related to the parotid gland. The best treatment for mixed tumor of the parotid gland is complete excision of the tumor; but great care must be exercised to save the facial nerve. Incomplete removal of a benign tumor is preferable to facial paralysis. Intracapsular removal of benign tumors may be justified in some cases, although recurrence of the tumor is to be expected in a large percentage of cases treated in this way. Mixed parotid tumors are rather insensitive to x-ray therapy, but postoperative radiation appears to lessen the number of recurrences. The parotid gland has a large superficial lobe, and a deep smaller lobe, joined by a narrow neck. The main trunks of the facial nerve do not penetrate the gland, but lie between these two lobes. During excision of a parotid tumor it may be necessary to dissect the nerve free from the parotid gland.

with Hodgkin's disease. After a considerable time there is usually spontaneous recovery. A biopsy must be taken to settle the diagnosis. The value of all treatments is doubtful, but x-ray therapy and nitrogen mustard may have some value. Half the cases have enlarged glands.

DISEASES OF THE SALIVARY GLANDS

Injuries

Accidental wounds or surgical operations may cause lacerations of the parotid gland, or wounding of the parotid duct. A radical dissection of the cervical glands usually makes it necessary to remove the lower portion of the parotid gland. Laceration of the gland may cause a discharge of saliva for several days, but the wound usually heals in a few days without persistent salivary fistula. A complete transection of Stenson's duct may continue as a salivary fistula, the discharge of saliva being more copious after eating. Pressure dressings, if instituted early, may aid in healing of the fistula. A fistula that persists after one or two months should be treated by operation, either to join the ends of the duct, or to transplant the proximal end of the duct into the mucous surface of the cheek. X-ray therapy will aid in reducing the secretion of saliva.

Inflammation

The parotid gland is more frequently affected by inflammation than are the other salivary glands.

Suppurative Parotitis occurs most often in debilitated, dehydrated patients, sometimes as a postoperative complication, or in prolonged febrile diseases like typhoid fever. Parotitis may, rarely, be associated with calculi in the duct or gland. Since intravenous salines and antibiotics have been in common use, acute parotitis has become a more infrequent postoperative complication. The local symptoms are pain and swelling of one or both parotid glands, and a foul dry

mouth. Pus may discharge from the red swelling about the opening of Stenson's duct. *General symptoms include fever, chills, headache, and general malaise.* There is usually a leukocytosis, and abscess formation may occur. Suppurative parotitis is considered to be a serious complication as it appears in patients already frail and ill.

To prevent this complication, the mouth should be kept clean, and dehydration must be avoided. Chewing of gum promotes salivation and good drainage of the gland. Antibiotics, intravenous fluid and mouth washes aid in treatment. Small doses of Lugol's solution may be useful. X-ray therapy will reduce secretion, and it will relieve pain and swelling. If an abscess forms, it must be incised.

Chronic Parotitis may follow an acute inflammation of the gland. Patients suffering from this disease may have recurrent or persistent swelling of the parotid gland. An injection of the duct with Lipiodol, a sialogram, may show a stricture of Stenson's duct, but narrowing of the duct is rare.

The mouth should be cleaned up, as the gland usually is involved by an ascending infection from the mouth. Dilatation of a narrowed duct is indicated. A stricture may be incised. X-ray therapy to reduce gland secretion offers the best chance of cure.

Mikulicz's disease is a chronic inflammatory ailment causing progressive enlargement of salivary and lacrimal glands. There is usually a proliferation of lymphoid and reticular cells with loss of salivary epithelium. X-ray therapy is the most satisfactory treatment.

Salivary Calculi

Calculi in Stenson's duct are rare, but they are not uncommon in the submaxillary (Wharton's) duct or gland. Calculi may cause recurrent inflammation and swelling in a submaxillary gland, aggravated by eating. X-rays of the gland may show the calculus. If the stone is in the duct, it may

CHAPTER XII

SURGERY OF THE THYROID AND PARATHYROID GLANDS

STEWART BAXTER, M.D.

THYROID GLAND

Embryology.—The major portion of the thyroid gland originates from the pharyngeal entoderm at the level of the first pouch. It soon develops into a solid mass attached to its point of origin by a narrow stalk—the thyroglossal duct. About the fifth week this stalk disappears leaving a small depression at the base of the tongue, known as the foramen cecum. The remainder of the stalk is often found as a fibrous cord but in some individuals remnants of epithelium lead to the formation of thyroglossal cysts and sinuses. The mass now assumes its position with a lobe on each side of the trachea and in the seventh week fuses with the rudimentary fifth pouches, which are ultimately transformed into thyroid tissue. The primitive mass now undergoes transformation into acini and follicles containing colloid material.

Anatomy.—In the adult, the thyroid gland, consisting of a right and a left lobe, joined by the isthmus, lies on the anterior surface of the larynx and trachea at the level of the fifth, sixth, and seventh cervical vertebrae. A third, or pyramidal lobe, often extends from the isthmus or medial aspect of either lateral lobe to the level of the hyoid bone. The average weight of the gland is 30 to 50 grams.

The arterial supply comes from:

1. The superior thyroid artery, a branch of the external carotid.
2. The inferior thyroid artery, a branch of thyrocervical trunk of the subclavian
3. The thyroidea ima artery which arises from the innominate or arch of the aorta.

The veins form a plexus that emerges as the superior and middle thyroid veins, which empty into the internal jugular vein. The inferior thyroid vein joins the innominate vein.

The gland is covered by a loose external capsule, a part of the deep cervical fascia, and an internal one bound to the gland forming the interlobular septa. The parenchyma is composed of groups of acini, or follicles lined by low cuboidal epithelium. The acini are normally filled with iodine containing colloid, which after fixation stains deeply with eosin. The important relations of the thyroid gland are the larynx and trachea which it partly surrounds; the carotid sheath, laterally; the esophagus posteriorly to the trachea, the recurrent laryngeal nerve, and the parathyroid bodies.

Physiology.—The thyroid is an important ductless gland, regulated by the thyrotropic hormone of the anterior lobe of the pituitary gland and has a close relationship to the sex glands. Its two main functions are:

1. To regulate body metabolism
2. To stimulate physical and mental growth

An overactive gland in adults produces a clinical state of hyperthyroidism or Graves' disease, whereas an underactive gland results in myxedema. In children an underactive gland is associated with a state of cretinism—dwarfed stature, underdeveloped secondary sex characteristics and marked mental retardation. The hypoactive state can be controlled by the administration of thyroxin, the

Malignant Tumors of the Parotid Gland

Cancers of the salivary glands are most common in the fifth decade. The malignant tumors tend to be stony hard and fixed. The outline is not discrete. They grow more rapidly than mixed tumors, but they progress more slowly than most cancers. Metastases, though late, may appear in the regional glands, lungs, bones, and distant organs. There may be severe pain, and facial paralysis is common in parotid cancers. Although tumors in the submaxillary and lacrimal glands are scarcer, a higher percentage of tumors in these locations is malignant.

Treatment.—If the cancer has not progressed too far, excision of the gland is indicated. In parotid cancers it is often necessary to sacrifice the facial nerve. The operation should be followed by x-ray therapy. Less than 50 % of the patients are free from recurrence five years after treatment by x-ray and excision.

REFERENCES

Face

- Ackerman, L. V., and del Regato, J. A.: *Cancer: Diagnosis, Treatment, and Prognosis*, St. Louis, 1947, The C. V. Mosby Company.
 Caylor, H. D.: Epitheliomas in Sebaceous Cysts, *Ann Surg* 82: 164-176, July, 1925.
 Figs, F. A.: Malignant Tumors of the Scalp, *S Clin North America* 26: 859-870, 1946.
 MacFee, W. F.: The Surgical Treatment of Large Hemangiomas of the Face in Children, *S Clin North America* 27: 431-442, 1947.

- Martin, Hayes E., MacComb, W. E., and Blady, J. V.: Cancer of the Lip, *Ann. Surg.* 114: 220-242, 341-368, Sept., 1941.
 Pohle, E. A., and McAneny, J. B.: Radium Treatment of Vascular Nevi: An Analysis of 152 Cases Seen During 1928-1938, *Am J Roentgenol* 44: 747-755, Nov., 1940.
 Richards, G. E.: The Treatment of Cancer of the Tongue, *Am. J. Roentgenol* 47: 191-206, 1942.

Neck

- Bailey, Hamilton: The Clinical Aspects of Branchial Fistulae, *Brit. J Surg* 21: 173-182, Oct., 1933.
 Casberg, M. A.: The Clinical Significance of the Cervical Fascial Planes, *S Clin North America* 30: 1415-1434, 1950.
 Harrington, S. W., Clagett, O. T., and Dockerty, M. B.: Tumors of Carotid Body, *Ann Surg* 114: 820-833, Nov., 1941.
 Labey, Frank: Tumors of the Neck, *S Clin North America* 27: 486-500, 1947.
 Martin, Hayes E.: The Treatment of Cervical Metastatic Cancer, *Ann. Surg* 114: 972-986, Dec., 1941.
 Slaughter, D. P.: Neck Dissections: Indications and Technique, *S Clin North America* 26: 102-115, 1946.

Salivary Glands

- Blady, J. V., and Hocker, A. F.: Sialography, Its Technic and Application in Roentgen Study of Neoplasms of the Parotid Gland, *Surg, Gynec & Obst* 67: 777-787, 1938.
 Clarke, T. H.: Parotid Gland Tumors, *S Clin North America* 32: 175-193, 1952.
 Kirklin, J. W., McDonald, J. R., Harrington, G. W., and New, G. B.: Parotid Tumors, *Surg, Gynec & Obst* 92: 721, 1951.
 McWhorter, G. L.: The Relations of the Superficial and Deep Lobes of the Parotid Gland to the Ducts and the Facial Nerve, *Anat Rec* 12: 149-154, 1947.
 Schulz, M. D., and Weisenberger, D.: The Sialogram in the Diagnosis of Swelling About the Salivary Glands, *S Clin North America* 27: 1156-1161, 1947.

clear or purulent mucoid material. If the sinus connects with the foramen cecum, clear saliva exudes.

The *differential diagnosis* includes submental lymphadenitis, dermoid or sebaceous cyst, ectopic thyroid or pyramidal lobe enlargement and tuberculous fistula.

The *treatment* is surgical removal of the cyst and tract following it to the foramen cecum by division of the hyoid bone if necessary. Injection of the cyst or tract with methylene blue prior to the dissection aids in tracing the sinus tract to its highest level. Unless completely removed, recurrence is inevitable. As in lingual thyroid, proof of the existence of a normal thyroid prior to excision of a cyst is essential.

Inflammation of the Thyroid Gland

ACUTE THYROIDITIS

Thyroiditis is a disease of unknown etiology. It may occur in a subacute form or as an acute suppurative process which goes on to a localized abscess requiring incision and drainage. The subacute form is the more frequent. The disease affects females twice as frequently as males and occurs chiefly in the 20 to 50 year age group. Upper respiratory infections are frequently a predisposing cause.

Signs and Symptoms.—There is usually a sudden onset of swelling and tenderness of one lobe of the thyroid gland, although the entire gland may be affected. Migration from one lobe to the other is common. There may or may not be fever, chills, cough, and dysphasia. The affected part of the gland is hard, tender, and the superficial tissues are frequently reddened and edematous if the process is situated anteriorly. The basal metabolic rate is not elevated and there are no eye signs of hyperthyroidism. Most cases run a self-limiting course of several weeks or months if untreated or if localization and suppuration do not supervene.

The *differential diagnosis* includes, hemorrhage into a degenerating colloid nodule,

carcinoma and phlegmon of the neck. In protracted and doubtful cases biopsy is indicated.

Various specific *treatments* have been advocated. Antibiotics do not affect the subacute type. X-ray therapy has brought about resolution of the process in many cases. The use of antithyroid drugs, propylthiouracil over weeks has also produced the same result. If a localized abscess occurs it should be incised and drained.

Pathologically, there is evidence of a diffuse subacute inflammatory process. Leukocytic infiltration and giant cells are noted.

CHRONIC THYROIDITIS

Two distinct forms of a chronic thyroiditis, struma lymphomatosa (Hashimoto's disease) and Riedel's struma (woody thyroiditis) are recognized.

Struma lymphomatosa is a chronic degenerative disease of the thyroid gland in which there are varying degrees of lymphoid and fibrous tissue replacement. It is not regarded as the end result of subacute thyroiditis nor is it associated with Riedel's struma. The etiology is unknown. It occurs chiefly in women in the 30 to 40 year age group.

Signs and Symptoms.—Firm symmetrical, or finely nodular enlargement of the gland is the chief symptom. It may be present for years. There are no associated toxic or febrile symptoms. Many cases show a moderate degree of hypothyroidism and occasionally myxedema. Large tumors encircling the trachea cause symptoms of compression. There is no tendency toward spontaneous remission.

The *diagnosis* is usually only made at operation, and cancer of the thyroid may be suspected. Recently Crile has advocated biopsy with the Silverman needle and mild doses of x-ray therapy.

Treatment.—Thyroidectomy is usually performed on the larger types. Conservative resection is advised, since subtotal or total resection leads to severe myxedema. Enough

active hormone of the gland or thyroid extract. Cholesterol metabolism is also influenced by the thyroid gland. Thus in hyperthyroidism the blood cholesterol is reduced, and in myxedema elevated.

single, in the midline, and occurs predominantly in females. The treatment consists in surgical removal provided that there is thyroid tissue in the normal site, otherwise total myxedema will result.

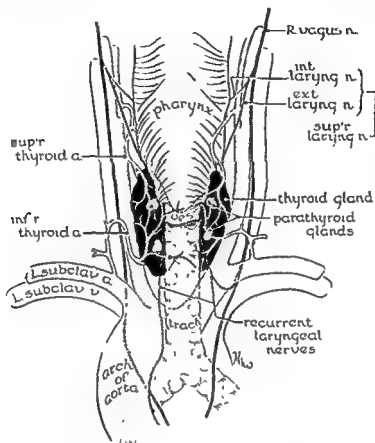


Fig 128—Anatomy of thyroid and parathyroid glands (posterior view).

Congenital Anomalies

LINGUAL THYROID

This anomaly represents the persistence of a part or all of the thyroid gland at its point of origin at the base of the tongue or foramen cecum. It is present from birth but tends to enlarge at puberty, during pregnancy, and at the menopause. Symptoms are due to the presence of a swelling at the base of the tongue, difficulty in speaking, swallowing or breathing, and a desire to swallow continuously. Bleeding may result from trauma and ulceration. The tumor is

THYROGLOSSAL CYSTS AND SINUSES

Signs and Symptoms—These anomalies may occur at any site along the course of the thyroglossal duct. They are most common below the hyoid bone, but may be found at any level from the foramen cecum to the sternal notch. They present in the midline as a firm, round swelling averaging 1 to 2 cm. in diameter. They are attached to the deep structures, particularly the hyoid bone but not to the skin unless infection has occurred. If a sinus is present, it is also in the midline and frequently results from incision and drainage of an infected cyst. It exudes

trophy is established it never completely regresses, and these goiters progress over a period of years to multinodular goiter and its complications. The universal use of iodized salt, the addition of small amounts of iodine 1 M Lugol's solution once a week in early cases will help to prevent subsequent enlargement.

NODULAR GOITER WITHOUT HYPERTHYROIDISM

This type of goiter results from previous adolescent enlargement and is the end result of endemic goiter. The complications of this type of goiter are (1) pressure symptoms from excessive growth—dyspnea, dysphagia, and hoarseness; (2) hyperthyroidism; (3) intrathoracic growth; (4) development of malignancy.

The treatment of multinodular goiter is somewhat controversial. In the early stages, surgery may not be indicated. However, when any of the above complications are present, as well as for cosmetic reasons, thyroidectomy should be performed.

THE SINGLE DISCRETE ADENOMA

Recently the existence of a discrete firm nodule in the thyroid gland has come to be regarded as a different and more serious problem, quite separate from that of multinodular goiter. The discrete adenoma has clinical and pathological possibilities that make its removal advisable regardless of size or other constitutional symptoms, for it is this type of nodule that shows the highest incidence of malignancy (20 %).

DIFFUSE GOITER WITH HYPERTHYROIDISM (GRAVES' DISEASE)

The exact etiology of toxic goiter is not definitely known. Certain theories have been formed. It is thought by many that overactivity of the thyroid gland is brought about by excessive stimulation by the thyrotrophic hormone of the anterior pituitary. The varying degrees of exophthalmus when present are regarded as due to this same source.

The use of tracer doses of radioactive iodine and the radioautograph of the gland have demonstrated important facts. By this method it has been shown that in many cases of solitary toxic adenoma, the adenoma is solely responsible for the toxicity, the remaining thyroid tissue being inactive.



Fig 129—Photograph of patient with exophthalmic goiter, showing exophthalmus and typical facies (From Meakins: Practice of Medicine, The C. V. Mosby Co.)

Severe mental strain, anxiety, shock, and acute illnesses have been regarded as predisposing factors. Females are affected three or four times as often as males. The 20 to 50 year age group includes the greatest majority of cases.

The signs and symptoms of hyperthyroidism are produced chiefly by the increased metabolic rate and increased oxygen consumption of the tissues. The objective signs are characteristic at first, but the symptoms have considerable variations and in the absence of objective signs are not reliable in diagnosis.

Nervousness, tremor, sweating, excessive appetite are common symptoms. The objective signs include tachycardia, capillary pulse,

gland is removed from the isthmus and lobes to relieve pressure on the trachea.

The *pathological picture* is characterized by extensive degeneration of the thyroid acini and replacement by lymphoid and fibrous tissue. Well-developed germinal centers are present. There is no extension to the capsule or surrounding tissues.

Riedel's Struma.—Riedel's thyroiditis is a *chronic inflammatory fibrosing process* involving one or both lobes of the thyroid as well as the surrounding capsule, muscles, trachea, and blood vessels. It produces a hard bulky tumor which may be indistinguishable from advanced cancer. It is a distinct clinical and pathological entity of which the etiology is unknown.

Signs and Symptoms.—The onset is slow, insidious, and not accompanied by pain or tenderness. Pressure symptoms are common, due to distortion or compression of the trachea. There is little systemic reaction, and the metabolic rate is normal except in advanced bilateral cases, when it is subnormal. The tumor is stony hard and fixed, leading to the presumptive diagnosis of cancer.

Treatment.—X-ray has no effect on this type of thyroiditis. Surgical removal is difficult due to fibrosis extending outside the gland. Enough gland should be removed to relieve pressure symptoms. Recurrence in the remaining lobe in unilateral cases has been reported. Radical removal is usually impossible, and the danger of injury to recurrent nerves, vessels, and parathyroid glands is increased.

Specific Thyroiditis.—Tuberculosis and syphilis of the thyroid gland are practically unknown.

Tumors of the Thyroid Gland

This classification for descriptive purposes is divided into (A) nonmalignant, and (B) malignant tumors.

(A) Nonmalignant Tumors

By far the most common tumor of the thyroid gland is described loosely by the

term *goiter*; other benign histoid tumors of the thyroid gland, such as fibroma, chondroma, myxoma, are so rare that no further elaboration is necessary.

Goiter, as a term applied to various types of enlargement of the thyroid gland, has been classified in various ways, none of which are entirely satisfactory. That adopted by the American Society for the Study of Goiter follows:

(A) Diffuse Goiter

- (1) without hyperthyroidism
- (2) with hyperthyroidism

(B) Nodular Goiter

- (1) without hyperthyroidism
- (2) with hyperthyroidism

In this classification diffuse goiter with hyperthyroidism corresponds to Graves' disease, and nodular goiter with hyperthyroidism to "toxic adenoma."

Diffuse and nodular goiter without hyperthyroidism represent the various manifestations of endemic or colloid goiter. In addition Crile uses the term "discrete adenoma" to describe a clinical entity, when there is an adenoma which from the clinical standpoint has the qualities of neoplasia rather than of an involutionary colloid nodule. This concept has some bearing on the higher incidence of carcinoma in single as opposed to multinodular goiters.

DIFFUSE GOITER WITHOUT HYPERTHYROIDISM

Etiology.—This corresponds to the simple adolescent colloid goiter—endemic goiter of iodine-deficient areas. It appears usually during puberty or pregnancy when the physiological demands on the gland are greatest. With recurring episodes of iodine deficiency, the gland begins to react irregularly and the multinodular involutionary type of goiter develops. This is the end result of many adolescent colloid goiters and accounts for the large nodular glands seen in older women in goiterous areas.

The *treatment* of adolescent goiter lies chiefly in its prevention, since once hyper-

trophy is established it never completely regresses, and these goiters progress over a period of years to multinodular goiter and its complications. The universal use of iodized salt, the addition of small amounts of iodine 1 m. Lugol's solution once a week in early cases will help to prevent subsequent enlargement.

NODULAR GOITER WITHOUT HYPERTHYROIDISM

This type of goiter results from previous adolescent enlargement and is the end result of endemic goiter. The complications of this type of goiter are (1) pressure symptoms from excessive growth—dyspnea, dysphagia, and hoarseness; (2) hyperthyroidism; (3) intrathoracic growth; (4) development of malignancy.

The treatment of multinodular goiter is somewhat controversial. In the early stages, surgery may not be indicated. However, when any of the above complications are present, as well as for cosmetic reasons, thyroidectomy should be performed.

THE SINGLE DISCRETE ADENOMA

Recently the existence of a discrete firm nodule in the thyroid gland has come to be regarded as a different and more serious problem, quite separate from that of multinodular goiter. The discrete adenoma has clinical and pathological possibilities that make its removal advisable regardless of size or other constitutional symptoms, for it is this type of nodule that shows the highest incidence of malignancy (20 %).

DIFFUSE GOITER WITH HYPERTHYROIDISM (GRAVES' DISEASE)

The exact *etiology* of toxic goiter is not definitely known. Certain theories have been formed. It is thought by many that overactivity of the thyroid gland is brought about by excessive stimulation by the thyrotrophic hormone of the anterior pituitary. The varying degrees of exophthalmus when present are regarded as due to this same source.

The use of tracer doses of radioactive iodine and the radioautograph of the gland have demonstrated important facts. By this method it has been shown that in many cases of solitary toxic adenoma, the adenoma is solely responsible for the toxicity, the remaining thyroid tissue being inactive.



Fig 129—Photograph of patient with exophthalmic goiter, showing exophthalmus and typical facies. (From Meakins' Practice of Medicine, The C. V. Mosby Co)

Severe mental strain, anxiety, shock, and acute illnesses have been regarded as predisposing factors. Females are affected three or four times as often as males. The 20 to 50 year age group includes the greatest majority of cases.

The signs and symptoms of hyperthyroidism are produced chiefly by the increased metabolic rate and increased oxygen consumption of the tissues. The objective signs are characteristic at first, but the symptoms have considerable variations and in the absence of objective signs are not reliable in diagnosis.

Nervousness, tremor, sweating, excessive appetite are common symptoms. The objective signs include tachycardia, capillary pulse,

high pulse pressure, loss of weight, diffuse enlargement of the thyroid gland and various eye signs. Palpable enlargement of the gland and exophthalmus are not always present. Diarrhea and a negative calcium balance, resulting in skeletal decalcification may be found in severe cases. Amenorrhea also occurs. Weakness of various groups of skeletal muscles may be a feature in some cases especially the quadriceps group. Carbohydrate metabolism is disturbed due to impaired glycogen storage in the liver, leading to glycosuria and elevated glucose tolerance curves, during the digestive phase.

Diagnosis — Symptomatically hyperthyroidism may be confused with anxiety states or neuroses, and in older individuals cardiac symptoms such as fibrillation or early decompensation may obscure the picture. The basal metabolic rate is the most reliable test of thyroid hyperactivity. Several tests may be required to obtain the true level. A lowered blood cholesterol level is a constant finding in most cases. Recently the determination of the protein bound blood iodine has been found to correspond with the degree of hyperthyroidism, but it is a difficult and expensive test. Occasionally a therapeutic test with iodine or an antithyroid drug will verify the diagnosis. Estimation of the uptake of a tracer dose of radioactive iodine has recently been used as a diagnostic test.

The treatment of hyperthyroidism has in recent years become more complicated. For many years the standard procedure was preoperative preparation with iodine and thyroidectomy. While this is a satisfactory procedure in many cases, the degree of improvement prior to operation was minimal in severely toxic debilitated patients, or those with cardiac complications. The discovery of the antithyroid drugs thiouracil and propylthiouracil in 1943 brought about a new era in the treatment of toxic goiter. With these drugs it is now possible to reduce a high metabolic rate to normal and keep it there for indefinite periods of time so that the patient comes to operation in the best possible physical condition.

In certain carefully selected mild cases, the prolonged use of these drugs may bring about a permanent remission.

Recently the use of radioactive iodine has opened a new and still experimental method of controlling and curing hyperthyroidism.

Choice of Treatment.—The decision as to the best form of treatment in cases of diffuse or nodular hyperthyroidism is based on a careful clinical, metabolic and social investigation of the individual case. While thyroidectomy after adequate preparation is still the treatment of choice in patients who are young and good surgical risks, there are definite indications for the other forms of treatment now available.

Indications for Definitive Medical Treatment With Antithyroid Drugs.

1. Cases with mild hyperthyroidism and small or moderate enlargement of the gland.
2. Recurrent hyperthyroidism following thyroidectomy.
3. Older age groups or those with some physical debility contraindicating surgery.
4. Certain cases who elect medical therapy. Propylthiouracil, 100 to 200 mg three times a day is given until the metabolic rate falls to normal or slightly subnormal. The dosage can then be reduced until the maintenance dose is found. Treatment usually must be continued for 1 to 2 years, at which time the drug is gradually withdrawn. Permanent remissions vary between 40 and 60 % in the milder cases. In moderately and severely toxic patients, with enlarged glands, the recurrence rate after discontinuing antithyroid drugs is high and this type of case does better with thyroidectomy.

Preparation for Thyroidectomy.—While the use of propylthiouracil has greatly displaced iodine in the preoperative preparation of patients for thyroidectomy, iodine is still advantageous as it allows good risk pa-

tients to come to operation in 10 days to 2 weeks as compared to the 4 to 8 weeks' interval required for propylthiouracil.

Indications for Thiouracil Preparation.

1. Severely toxic cases with large goiters, diffuse or nodular.
2. Cases with cardiac complications.
3. Cases where the gland enlarged under medical treatment.

The toxicity of propylthiouracil is slight as compared to thiouracil, but the rare case of leukopenia and agranulocytosis has been reported. The appearance of any unusual symptoms such as a rash, glandular enlargement, diarrhea, calls for immediate withdrawal of the drug and careful reassessment of the blood picture. Iodine is always given for a week or 10 days prior to operation to reduce vascularity and friability of the gland which may be a technical difficulty when thiouracil alone is used.

Indications for the Use of Iodine Alone in Preoperative Preparation.

1. Young good risk patients with diffuse or nodular goiter and milder type of hyperthyroidism who do not wish to undergo the uncertainties of prolonged medical treatment.
2. Any case in which toxicity to antithyroid drugs develops.

Iodine is administered as Lugol's solution 5-10 ml 3 times a day with meals after clinical assessment of the patient. The patient should be in hospital; the basal metabolic rate is checked every 2 to 3 days. At the height of clinical improvement, operation should be performed immediately since many cases will escape iodine control and toxicity will return. The average length of the time is 10 days to 2 weeks.

Indications for Use of Radioactive Iodine.

The use of radioactive iodine has increased in recent years with the greater availability of radioactive isotopes. Encouraging results are reported from various centers. The drug is given in water, either one estimated total dose or multiple small doses of 5-10 milli-

curies at intervals. The incidence of myxedema seems to be higher with the single dose technique. The multiple dose method requires about 6 months to secure control of hyperthyroidism. The possibility of late effects of gamma radiation on the thyroid gland are not yet fully known. Its use is restricted at present to

1. Older age groups with short life expectancy.
2. Recurrent cases that are poor surgical risks.
3. Patients who refuse surgery and do not respond to antithyroid drug treatment.

X-ray Therapy has been used in certain cases of recurrent hyperthyroidism when surgery is not indicated and antithyroid drugs are ineffectual in controlling toxicity. Prolonged treatment is necessary to control hyperthyroidism and radioactive iodine if available is a preferable method for handling such cases.

Thyroidectomy

Preoperative Treatment.—During the administration of iodine or antithyroid drugs, careful attention must be given to other aspects of hyperthyroidism. Adequate rest or bed rest with bathroom privileges is required. A high caloric, high vitamin diet is essential to restore lost weight. Extra carbohydrates are necessary to replace glycogen depletion of the liver. Sedation, bromides, and barbiturates are useful in controlling nervous manifestations. Patients with incipient decompensation or auricular fibrillation should be digitalized prior to operation.

Anesthesia for Thyroidectomy.—The early technique of local infiltration and cervical block has largely disappeared in favor of newer anesthetic agents. The administration of a basal anesthetic such as Pentothal or Avertin in the patient's room followed by intubation and endotracheal nitrous oxide or cyclopropane anesthesia is the ideal combination. It assures an unobstructed airway at all times and reduces the total amount of anesthetic agent required.

The technique of thyroidectomy is now well standardized and emphasizes the following points: (1) a well-controlled properly prepared patient; (2) anatomic dissection of the gland, (3) exposure and protection of the recurrent laryngeal nerves; (4) preser-

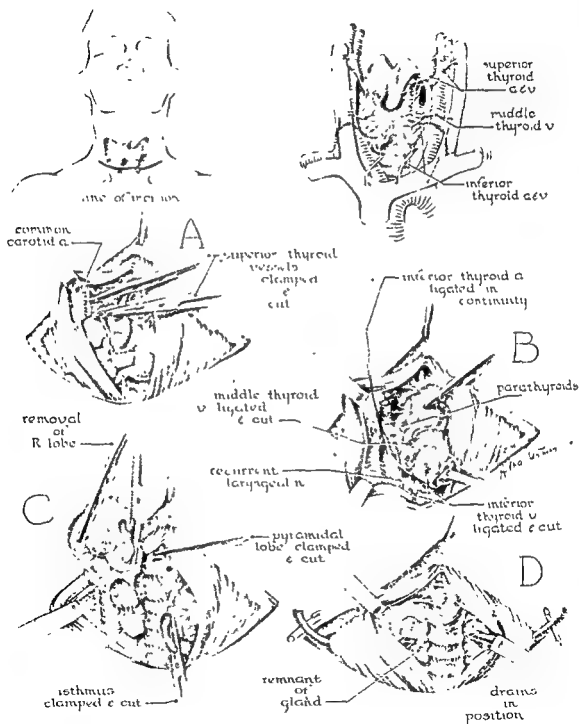


Fig 130—Technique of the stages of thyroidectomy

vation of the parathyroid bodies; (5) resection of the optimum amount of gland to effect a cure but not to produce myxedema; (6) careful hemostasis and good cosmetic closure of the incision.

A low collar incision is placed 1 to 1½ inches above the sternal notch. The superior thyroid artery is then isolated, clamped, and doubly ligated with silk or heavy catgut, care being exercised not to injure the superior laryngeal nerve.

The middle thyroid and inferior thyroid veins are now divided and ligated, and the gland is rotated medially to expose the posterior capsule. The inferior thyroid artery is identified and ligated well away from the capsule. The parathyroid bodies may occasionally be identified close to the entrance of the branches of the inferior thyroid artery into the gland. Careful dissection in the region of the branches of the inferior thyroid artery exposes the recurrent laryngeal nerve as it approaches the gland and reduces the incidence of nerve damage. The gland is now divided across the isthmus and the lobe is resected from the midline outward, leaving the posterior capsule intact over the recurrent nerve and the parathyroid bodies. The amount of tissue left is largely an individual factor. Usually it represents only a thin layer of tissue in the tracheo-esophageal angle and on the posterior capsule. The isthmus and pyramidal lobe, if present, should be removed. The opposite lobe is similarly resected, careful hemostasis is secured; drains are placed to either thyroid bed emerging behind the sternomastoid muscles in the line of incision. The cervical fascia and thyroid muscles are sutured and the skin is approximated carefully with clips.

The complications of thyroidectomy are classified as follows. I Early. II Late.

I. Early.

1 *Postoperative shock* is prevented by the use of adequate amounts of glucose solution and blood or plasma if indicated.

2. *Postoperative hemorrhage.* Postoperative bleeding will occur occasionally even after careful attention to hemostasis. If a large enough clot develops, it will eventually cause sufficient pressure on the trachea and larynx to produce dyspnea and stridor. If these symptoms are not relieved, the patient will die from asphyxia. Inspection of the wound, and if bulging and tense, removal of clips and opening of deep cervical fascia may be a lifesaving procedure. Later evacuation of the clot and ligation of the bleeding point can be done in the operating room. If the laryngeal compression has been present for some hours before release, a temporary tracheotomy may be required to provide an adequate airway since edema and swelling of the true and false cords may be marked enough to obstruct respiration.

3. *Recurrent nerve injury.* Partial or complete injury to one recurrent laryngeal nerve does not as a rule give any serious respiratory embarrassment. There will be difficulty in expectoration of mucus. The voice will be hoarse but ultimately will return more or less to normal. Section of both nerves produces complete obstruction of the airway since both cords fall into the cadaveric position. Serious dyspnea, cyanosis, and stridor develop soon after operation and require lifesaving tracheotomy.

4. *Tetany* results from removal of parathyroid bodies and may be temporary or permanent. It usually manifests itself about the third or fourth postoperative day with numbness and tingling, carpopedal spasm, and positive Chvostek's sign. The administration of calcium gluconate intravenously will relieve any alarming symptoms. The blood calcium is usually below 8 mg %. The majority of cases are transitory and yield to symptomatic treatment. Permanent tetany requires the use of A. T. 10 dihydrotachysterol, and increased calcium ingestion.

5. *Thyroid crisis.* This is an increase in all the preoperative symptoms, tachycardia, hyperthermia, extreme restlessness, delirium

and coma, which may appear during the first 24 to 48 hours postoperatively. It occurs rarely, and less frequently since the use of the antithyroid drugs. The treatment consists in heavy sedation morphine gr. $\frac{1}{4}$ to $\frac{1}{2}$ or even Sodium Pentothal intravenously and copious intravenous fluids 3,000 to 5,000 c c of 5 % glucose solution daily. Lugol's solution 20 to 60 minims may be added to the intravenous once or twice a day. The hyperthermia is controlled by ice water or alcohol sponges or the patient may be literally packed in ice. Death in delirium and coma rarely results.

Late —

1 Recurrent hyperthyroidism as distinguished from continuing hyperthyroidism occurs in about 2 % of thyroidectomies for toxic goiter. Years may elapse before the recurrence.

2 Myxedema or hypothyroidism is seen in temporary or permanent form in 3.5 % of thyroidectomies. It yields readily to the administration of thyroid extract and does not constitute a disability.

NODULAR GOITER WITH HYPERTHYROIDISM

Hyperthyroidism associated with nodular goiter occurs most often in older age groups from 40 to 60 years. There is little difference in the signs and symptoms and those of Graves' disease. There is a higher incidence of cardiac complications, auricular fibrillation, and early decompensation, due partly to the advanced age group and partly to the chronicity of many of these cases. The toxicity may be undiagnosed until cardiac symptoms supervene. The same principles apply to the management of this type of goiter as those laid down for diffuse toxic goiter. It is usually unwise to attempt definitive medical treatment with antithyroid drugs, and thyroidectomy after suitable preparation is indicated.

Malignant Tumors of the Thyroid Gland

Carcinoma of the thyroid gland is the most common form of malignant tumor en-

countered, but rare cases of sarcoma have been reported. Metastatic involvement of the thyroid gland is also very rare.

Carcinoma of the Thyroid Gland

The etiology of cancer of the thyroid is unknown. The presence of a preexisting nodule has been reported in as high as 50 % of cases. Diffuse hyperplasia rarely results in carcinoma.

The antithyroid drugs are carcinogenic in animals in large doses, and reports of carcinoma developing in cases of toxic goiter receiving prolonged thiouracil therapy have been recorded.

Cancer of the thyroid is more common in goiterous districts, affects females 3 to 4 times as frequently as males, and occurs most often in the 40 to 70 year age group.

Classification.

The various types of cancer of the thyroid differ markedly in their degree of malignancy and hence their prognosis.

A classification on this basis is the most satisfactory.

- I Tumors of low grade malignancy.
 - (a) Papillary cystadenoma
 - (b) Hurthle's cell tumor.
- II. Tumors of moderate malignancy
 - (a) Papillary adenocarcinoma
- III. Tumors of high grade malignancy.
 - (a) Adenocarcinoma.
 - (b) Giant cell carcinoma.
 - (c) Spindle cell carcinoma

The earliest symptom is the presence of a nodule in the thyroid gland which suddenly and progressively enlarges and becomes firmer than the surrounding gland. Nodularity and calcification may be noted. The appearance of dyspnea, dysphagia, pain, voice changes, loss of weight, and fixation of the mass to surrounding structures are all late manifestations and represent usually an inoperable or incurable stage of the disease. About 15 to 20 % of cases may show varying degrees of toxicity.

The clinical diagnosis of carcinoma of the thyroid is a dubious one and if obvious usu-

ally represents an inoperable stage. The diagnosis is unsuspected in about 40 to 50 % of cases of surgically removed nodules, and is only made on careful pathological study; 10 % of surgically removed nodular goiters and 24 % of solitary nodular goiters have been reported carcinomatous. Careful scrutiny of all nodular goiters and exploration of all suspicious cases, especially the solitary nodules is clearly indicated.

The treatment is radical thyroidectomy, but the technique is dictated by the individual case and the type of lesion suspected. In those cases where a subtotal thyroidectomy has been performed and the diagnosis made only on histological examination, if the focus is small and completely removed, the prognosis is good. Postoperative x-ray therapy gives added protection. In the papillary type of carcinoma, if glandular metastases are present, a careful dissection of the region involved is performed. X-ray therapy postoperatively is usually indicated.

In treating the more malignant types of carcinoma, ligation and resection of the internal jugular vein well above the limits of the gland are performed before removal of the thyroid lobe on the affected side is undertaken. Similarly the inferior thyroid veins are ligated early. This is done to prevent blood stream contamination during operation. The strap muscles and recurrent laryngeal nerve are sacrificed if involved by the tumor. Postoperative radiation is essential. Bilateral involvement by carcinoma is rarely operable. Frankly inoperable cases may be temporarily improved by heavy roentgen therapy usually combined with tracheotomy.

Radioactive iodine has been used in the treatment of carcinoma of the thyroid. Unfortunately most carcinomas of the thyroid are nontoxic and do not take up radioactive iodine in sufficient quantity to be of any therapeutic value.

The rare cases with toxicity have been successfully controlled with large doses of radioactive iodine over a long period of time (250 millicuries over a three-year period).

The use of tracer doses and estimation of the uptake by the involved gland or metastatic lesions indicate the possibility of treatment by this method.

The use of antithyroid drugs has been found to increase the absorption of radioactive iodine in certain cases.

Total thyroidectomy has been performed and causes an increased uptake of radioactive iodine by the metastatic lesions.

Radioactive iodine has not proved to be of significant value for the treatment of carcinoma of the thyroid gland.

SURGERY OF THE PARATHYROID GLANDS

Surgery of the parathyroid glands involves chiefly the treatment of hyperparathyroidism, which is caused by an adenomatous enlargement of one of the glands.

Embryology.—The parathyroid glands first appear as thickenings of the third and fourth pharyngeal pouches in the 10 mm embryo. Those arising from the third pouch remain attached to the thymic body, descend with it, and eventually lie on the posterior surfaces of the inferior poles of the thyroid. The other two glands remain in their original positions and are related to the posterior surfaces of the superior poles of the thyroid gland. This mode of origin accounts for the variability in the number and position of the parathyroid glands.

Physiology.—The function of the parathyroid glands is to regulate calcium metabolism, through their internal secretion, parathormone, which was isolated by Collip in 1925. Surgical removal of the parathyroids leads to a state of tetany. Overactivity of the parathyroids due to hyperplasia or adenoma results in the clinical syndrome of hyperparathyroidism.

Hyperparathyroidism

This disease was known for many years as von Recklinghausen's disease of bone or osteitis fibrosa cystica until 1925 when Mandl demonstrated the exact relationship

between the clinical syndrome and parathyroid adenoma.

Etiology—The cause of this condition in the great majority of cases is due to adenoma of a parathyroid gland, but in occasional instances there is hyperplasia of all parathyroid glands. The symptomatology is the same in either case. It occurs most commonly in the 20 to 40 year age group.

Signs and symptoms may be divided into three separate groups:

- 1 Those due to *increased blood calcium*. The blood calcium is always elevated, 12-18 mg % and the blood phosphorus reduced below normal (1-2 mg. %). This results in lassitude, hypotonia, weakness, constipation, anorexia, and loss of energy.
- 2 **Skeletal Involvement.** The excessive mobilization of calcium from the bones results in marked skeletal decalcification as evidenced by x-ray. Spontaneous fractures are common. Bone cysts and tumors which may be single, but usually multiple, are found on x-ray survey of the skeletal system.
- 3 **Symptoms of Renal Involvement.** Due to excessive urinary excretion of calcium and phosphorus, calculi, or calcinosis of the renal parenchyma, may occur. Signs of renal colic and renal infection are common.

The **diagnosis** may be difficult due to the variability of the signs and symptoms and simulation of other diseases. However, since the clinical entity has become better known, early diagnosis is less difficult. The x-ray and blood pictures are characteristic and usually diagnostic. The performance of a calcium balance study indicates that the patient is in markedly negative balance.

Rarely the tumor may be palpable in the neck or there may be forward displacement of the lateral thyroid lobe.

The **treatment** of hyperparathyroidism is exploration of the posterior surface of the thyroid gland and removal of the adenoma

or resection of two or three glands if hyperplasia is found. Considerable difficulty may be encountered if the adenoma is situated anomalously; cases requiring three, four, and five operations are reported.

The most common site for adenomas are at either lower pole (35 % each), but they may be found in the carotid sheath, behind the esophagus, and in the superior mediastinum.

Careful anatomical dissection must be maintained until the tumor is found. Frozen section of suspicious nodules removed at operation is a great aid in finding the adenoma, since the histological picture is quite characteristic.

Complications—Injury to recurrent laryngeal nerves is best avoided by careful dissection and identification of these structures early in the operation.

Postoperative tetany due to a sudden drop in blood calcium levels must be anticipated. Frequent determination of the blood calcium during the first postoperative days and the use of calcium gluconate intravenously and by mouth will usually control the hypocalcemia until the remaining glands stabilize the blood level.

A. T. 10, dihydrotachysterol, also aids in maintaining normal blood calcium levels over long periods of time.

Results.—Complete recalcification of the skeletal system occurs in about a year following removal of a parathyroid adenoma.

REFERENCES

- Astwood, E. B.: Thiouracil Treatment in Hyperthyroidism, *J. Clin. Endocrinol.* 4: 229-248, 1944.
- Crisle, George, Jr.: *Practical Aspects of Thyroid Disease*, Philadelphia, 1950, W. B. Saunders Company.
- Gordon, E. S., and Albright, E. C.: Treatment of Thyrotoxicosis With Radioactive Iodine, *J. A. M. A.* 143: 1129-1132, 1950.
- Means, J. H.: *The Thyroid and Its Diseases*, ed. 2, Philadelphia, 1948, J. B. Lippincott Company.
- Rienhoff, W. F., Jr.: The Surgical Treatment of Hyperparathyroidism, *Ann. Surg.* 131: 917-944, 1950.
- Surgical Practice of the Lahey Clinic*, by Members of the Staff, Philadelphia, 1951, W. B. Saunders Company.

CHAPTER XIII

THE BREAST

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Breasts are characteristic of the mammalian class of animals. Their function is to secrete milk for the nutrition of the young; but in addition as a secondary female sexual development, they serve, in the human at least, as a powerful attracting stimulus to the male. They are composed of epithelial elements, which are surrounded and supported by fibrous and fatty connective tissue—the stroma. In the active state the epithelial elements form compound tubulo-alveolar exocrine glands. In the resting phase no alveoli are present, they do not secrete and thus they cannot be considered true glands.

Embryology.—The remarkable thing is that, while the breasts are the last glands to function in the adult, they are the first of the epidermal glands to appear in the fetus. The glandular part of the breast is derived from ectoderm, which grows inward in strandlike fashion. The supporting stroma is evolved from mesodermal subcutaneous tissue. As early as the sixth week, an ectodermal thickening or outgrowth is apparent on either side of the trunk. In the second stage this milk line atrophies except in the pectoral region. This subsequently grows inward branching radially into 15 to 20 solid cords. These ectodermal cords eventually become the milk ducts and each represents a unit of breast tissue. They branch and subdivide and at 7 months begin to canalize. At birth the depression from the epithelial ingrowth becomes erected to form the nipple.

Anatomy.—The size, shape, and structure of breasts are extremely variable. The breast is situated, except for the prolongation of the tail, in the superficial fascia on the front of the thorax. From the sternum it extends to the midaxillary line and verti-

cally from the second to the sixth ribs. There is no capsule and the 15 to 20 radiating lobes are firmly imbedded in the superficial fascia by strands of fibrous tissue. These form the framework of the organ and are known as the ligaments of Cooper. They ramify throughout the breast substance and connect the skin to the deep fascia. These ligaments are very important from the standpoint of the diagnosis of malignant disease, because, when involved with carcinoma, they cause fixation and dimpling of the skin.

Each lobe of the breast is an independent gland composed of lobules. A lobule is made up of branching lobular ducts, the smallest of which are termed ductules. The ultimate terminations of the ductules are the secreting alveoli. The stroma immediately surrounding the smallest ducts and acini is termed the periductal or intralobular connective tissue and is a relatively cellular type of tissue subject to constant variations. The stroma associated with the nipple, lactiferous and larger lobular ducts is much denser since it is not subject to great change. This is termed the intralobar connective tissue. The fascia between the lobules is designated interlobular while that separating the lobes is termed the interlobar connective tissue.

Relations.—The gland lies on the pectoral fascia. Posterior to the upper half is the pectoralis major muscle, while inferiorly are the serratus anterior, the external abdominal oblique, and upper part of the rectus sheath. The axillary tail (of Spence) is particularly variable in size and extent. It passes upward and laterally at the edge of the pectoralis major and on through the deep fascia into the axilla.

Blood Supply.—The anterior perforating branches of the internal mammary artery,

particularly the second, third, and fourth, supply the medial portion. The lateral aspect is supplied by the lateral thoracic artery from the second part of the axillary as well as the lateral perforating branches of the corresponding intercostals. A point of surgical importance is the fact that the main arterial inflow is through the upper half of the breast. The venous drainage in general follows the course of the arteries. Enlarged bluish veins characteristic of pregnancy and lactation are often seen running independently in the subcutaneous tissues.

Lymphatics.—Detailed knowledge of lymphatic drainage of the breast is important because the aim of cancer treatment is not only the removal of the breast itself but also, all the associated lymphatic vessels and glands.

The lymphatic system of the breast originates in the interlobular spaces and on the walls of the lactiferous ducts, and terminates in nodes in the axilla, thorax, and cervical region.

(A) *Superficial*.—The plexuses of small lymph vessels, which surround the acini, pass via the periductal lymphatics to the subareolar plexus. From here efferent channels lead principally to anterior pectoral nodes along the lateral thoracic vein at the edge of the pectoralis major muscle.

(B) *Deep*.—Some vessels leaving the interlobular spaces and ducts pass posteriorly to the deep fascial plexus in relation to the pectoral and serratus muscles. From the deep plexus large lymphatic channels run to the regional lymph glands. The efferent vessels from this fascial plexus can be traced in groups circumferentially like the spokes of a wheel.

1. Some vessels pass directly above, after perforating the pectoralis major muscle and terminate in Rotter's and other interpectoral glands, which lie in relation to the pectoralis minor muscle. Efferents from here proceed to the subclavian nodes

2. From the medial aspect of the deep plexus vessels pass inward, alongside the perforating arteries, and enter the anterior mediastinal glands.

3. Some vessels from the deep plexus cross the midline and anastomose with the corresponding plexus of the other breast.

4. From the inferomedial part of the plexus, vessels travel through the rectus fascia to the ligamentum teres and thence to the liver and subdiaphragmatic lymph nodes.

5. On the lateral aspect, there are channels which pass inward with the intercostal blood vessels to terminate in a series of glands which lie inside the lateral chest wall and also to other glands in proximity to the aorta. This route is probably responsible for many of the vertebral metastases so frequently seen in breast cancer.

6. By far, the most important lymphatic drainage of the deep plexus is upward and laterally to the lateral pectoral nodes and thence to the axillary groups. Direct connections exist, however, from the breast to the axillary and subclavian nodes.

The lymph nodes of the axilla lie in relation to the axillary vein and its immediate branches, inside the deep fascia. Five groups are usually described.

1. *Apical Group*.—This is the largest and may contain as many as a dozen nodes. The apical group is situated at the apex of the axilla behind and proximal to the insertion of the pectoralis minor.

2. *Central Group*.—This group of nodes lies on the axillary floor. The palpability of this group depends on the thickness of the fascia and the amount of fat present.

3. *Posterior Group* (or subscapular).—This group is centered between the subscapularis and teres major muscles in relation to the subscapular vessels.

4. *Lateral Nodes*.—This group is situated medial to the coracobrachialis and short head of the biceps muscles in relation to the third part of the axillary artery.

5. *Anterior (Pectoral) Nodes*.—This group is included because they extend upward



ANTERIOR DISSECTION

SAGITTAL SECTION

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along the outer border of the pectoralis minor to the anterior wall of the axilla in relation to the lateral thoracic vessels.

All of these groups must be specifically sought for in operations for cancer. They drain through efferents to the subclavicular, supraclavicular, or cervical glands, and thence to the mediastinum, where they eventually empty into the thoracic or accessory thoracic duct.

Nerve Supply.—The skin covering the breast is richly supplied with branches of 4th, 5th, 6th intercostal nerves. The glandular part receives autonomic fibers which run with the 2nd to 6th intercostal nerves. Smooth muscle fibers which erect the nipple are supplied by these autonomic nerves.

Histology.—The alveoli are lined by cuboidal or columnar secreting epithelial cells which rest on the membrana propria in orderly fashion. During lactation, the mechanism of milk secretion involves constant exfoliation of these cells. Columnar epithelium lines the ducts as far as the lactiferous sinuses and then it becomes squamous in character. In the ducts a reserve outer layer of low cuboidal cells supports the columnar epithelium. The stroma consists of fat and fibrous tissue and is contiguous with the surrounding subcutaneous structures since the breast has no limiting capsule. Around the acini are so-called "accommodative" spaces consisting of loosely arranged connective tissue. Because the bulk of the breast is fat, the actual size bears no relation to the quantity of milk-producing structures. The normal male breast does not significantly alter throughout life but consists merely of a few small duct rudiments posterior to the vestigial nipple.

Physiology.—Most breast derangements are in some way related to the varying cellular characteristics that result from constantly changing hormonal stimulation. A more detailed knowledge of breast physiology than space permits here should be acquired. With the onset of puberty, the female breast

begins to undergo alternate waves of hyperplasia and involution. The hyperplasia is characterized by a material increase in both the duct structures and stroma. In the cycles of this process, which are coordinated with the menstrual periods, the phases of hyperplasia exceed the following involution phases until such time as the breast assumes the adult size. When pregnancy occurs there is a tremendous wave of proliferative activity and thousands of alveoli develop. In the latter stages of lactation, regressive changes occur in which these racemose glands undergo atrophy, and the acini disappear. Finally with the menopause there is the onset of a gradual reduction in both parenchyma and stroma, until eventually nothing remains but a few dilated ducts and a small amount of fatty fibrous connective tissue.

CONGENITAL ANOMALIES

Amazia

Complete absence of a breast is rare and is usually found in males. It may be associated with the absence of the pectoral muscles or other congenital aberrations.

Athelia and Polythelia

Athelia is a rare condition denoting absence of the nipple, whereas polythelia which is more common refers to the presence of accessory nipples without breast tissue. Accessory breasts may be found anywhere along the mammalian "milk-line" which extends from the axilla to the groin. Tumors can arise in these accumulations of breast tissue. Thus a primary carcinoma is occasionally seen in the axilla.

Gynecomastia

Gynecomastia is a true hypertrophy of mammary tissue in the male. The cause is usually unknown, although the condition is commonly seen in men undergoing estrogenic treatment for carcinoma of the prostate. It is also seen associated with hor-

hormone-producing tumors such as adrenocortical carcinoma, embryonal carcinoma, and especially chorionepithelioma. Testicular biopsies and blood estrogen determinations are often indicated and are occasionally of diagnostic significance in these cases. In the male excision of such breast tissue may be warranted to correct discomfort or to alleviate embarrassment. Painful enlargements of the breasts are sometimes seen in males at puberty. Such lesions should be treated conservatively, since they are due to a temporary hormone disturbance. The effects of hormonal stimulation through the placenta is sometimes seen in the infant at birth. This is designated "mastitis neonatorum." The discharge known as "witch's milk" has given rise in the past to superstitious phantasies, as the involved baby is usually the first born and beyond term.

Virginal Hypertrophy (Macromastia)

This type of diffuse enlargement is usually unilateral. The cause is unknown. An abnormal breast of this nature seldom functions efficiently and is particularly prone to structural disease. Surgery may be indicated to alleviate embarrassment in extreme degrees of enlargement. This consists of intricate plastic procedures to reduce the size, or preferably, simple mastectomy.

INFLAMMATORY LESIONS

Acute Infections

Acute breast infections may occur at any period of life but are usually associated with lactation. With modern methods of prevention and management, infections have relinquished the important and frequent place they formerly held in breast surgery.

The causative organisms should always be identified for proper antibiotic therapy. *Staphylococcus aureus* and *Streptococcus hemolyticus* are the common offenders. The bacteria invade the breast through a crack in the nipple and pass inward by way of the ducts or the periductal lymphatics. A dif-

fuse cellulitis is first established, but continuation of the infective process leads to localization and suppuration.

Three varieties of abscess formation are formally described, depending on location.

1 **Premammary Abscess** may be subareolar or subcutaneous in position. Because of the location, there is no difficulty in the diagnosis. The classical signs of inflammation are present and the localized pus may be fluctuant. Prompt response to chemotherapy may be expected. The pus should be aspirated and the cavity filled with the antibiotic of choice. When well-advanced, the abscess should be radially incised under general or local anesthesia, the pus drained, and the cavity packed with gauze.

2 **Intramammary Abscess** is the usual type of acute breast infection and is located deep in the substance of the breast tissue. It practically always results from lactational mastitis. The cavity is diffusely loculated and may involve one or more lobes. There is marked systemic reaction with fever and malaise. The localized inflammatory reaction is associated with marked induration of the breast. In the early stages, the abscess cavity should be aspirated and the pus replaced with the correct antibiotic. If the abscess is well developed, operation under general anesthesia is indicated. A large radial incision into the abscess cavity is made and a finger is inserted thoroughly to break down the honeycomb type loculations in order to insure complete drainage. The cavity is then packed with gauze and the incision is left wide open. Dependent drainage with soft rubber tubes through a separate stab wound is often advisable.

3 **Retromammary Abscess** is a localized infection in the areolar tissue between the chest wall and the breast. It is extremely rare and may be acute or chronic. The acute cases are typified by a much more intense systemic reaction with less prominence of the local signs. Such infections are usually secondary to subjacent lesions, such as empyema, tuberculous ribs, or infected hem-

atomas. The typical abscess eventually points inferolaterally, pushing the breast forward. Treatment consists of surgical drainage in addition to correction of the underlying lesion. A curved incision is made along the inferolateral aspect of the breast. The abscess is sought for with a pair of artery forceps and digitally explored. Gauze is then packed into the cavity and any underlying lesion is dealt with.

application of a firm protective breast binder and 5 mg. of Stilbestrol 3 times daily will adequately serve this purpose.

Chronic Infections

Tuberculosis, while rare, is responsible for the majority of chronic infections. It is usually secondary but may be primary. A large tender mass of variable consistency is present and it may be absolutely indistinguish-

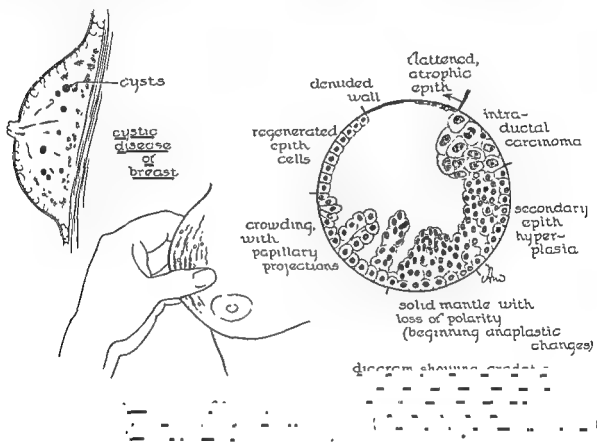


Fig 131—Chronic cystic disease of the breast

The important point in the treatment of all breast infections is prophylaxis by careful hygiene. This includes cleanliness of the nipple, protection from trauma, and prevention of stasis by expression of the preliminary secretion. The use of antibiotics at the time of childbirth has been dramatically effective in reducing the incidence of these infections. If an acute infection occurs, the baby should be weaned, and the breast "dried up." The

able from carcinoma. Certainly in the early stages, the clinical diagnosis is impossible. The axillary glands are characteristically enlarged and as the lesion advances multiple draining sinuses appear. The local treatment is mastectomy, but, in addition, the general treatment of tuberculosis must be enforced, such as rest, diet, dihydrostreptomycin, P. A. S. and other agents for which the future holds promise.

Syphilis of the breast is now a curiosity. As a primary lesion it appears as a chancre in the region of the nipple. It is single, unilateral, painless, and is associated with rapid enlargement of the axillary lymph nodes. Diagnosis is made by the typical appearance and by darkfield examination of exudate. In doubtful cases biopsy is necessary. The treatment is that of syphilis in general.

Actinomycosis is an extremely rare infection and is usually secondary to involvement of the lungs with extension through the thoracic wall. It is characterized by chronically indurated areas and weeping granulation tissue which discharges sulphur granules through secondarily infected sinuses. Treatment is surgical excision of the lesions, massive doses of iodides, penicillin, and other forms of chemotherapy.

CYSTIC DISEASE

The female breast is apt to be altered in structure first by fibrous involution with loss of parenchyma and second by varying degrees of epithelial hyperplasia. These two types of changes often coexist in varying degrees and as far as is known are due to abnormal hormonal stimulations. When such structural changes are evident, we call the lesion cystic disease, or fibroadenosis, for lack of a better term. Despite the fact that the varied terminology so commonly encountered in reading descriptions of the different phases or degrees of this type of breast change is extremely confusing, it is often possible to systematically follow the advancement of the abnormalities in structure in a more or less orderly progression. Whether or not it is customary for cancer to evolve from the associated series of epithelial changes is still a much debated point. Until this is settled the problem of properly treating the lesions called cystic disease remains difficult.

The fundamental changes consist of an increase in fibrous connective tissue with loss of parenchyma (epithelial elements). Dilatations of the remaining ducts due to ob-

struction from inspissated material, or extrinsic pressure may form cysts. Such cysts may reach considerable size and contain serous fluid under painful tension. They may be lined (in chronological order) by compressed atrophic epithelium, a denuded fibrous membrane, or by regenerated epithelium. The newly formed or regenerated epithelial cells are at first well orientated and highly differentiated (*primary epithelial hyperplasia*). Later, they may, due to crowding, develop papilliferous projections into the lumina. Further proliferative activity may result in the epithelial cells piling up, losing their orientation, and becoming anaplastic. They may form several layers lining the cysts and ducts, bridge across the lumina or solidly fill them. When this occurs it is referred to at this hospital as *secondary epithelial hyperplasia*. This is considered by some authorities as a lesion which may later develop into cancer. Further degrees of anaplasia and epithelial differentiation within the ducts may occur until, from the morphological point of view, the cells are considered malignant and the so-called *intraductal carcinoma* or *carcinoma-in-situ* is developed. As the diagnosis here depends on cytological changes alone, the borderline of malignancy cannot be sharply drawn. However, once these cells "break the law," i.e., the growth breaks through its normal boundaries and extends into periductal tissue or lymphatics, carcinoma is readily diagnosed on a topographical basis.

There is evidence to support the interrelation of the epithelial activity associated with cystic disease and the development of malignancy. Experimentally, in male mice, a similar series of epithelial changes, which progress to malignancy, have been reproduced by the injection of estrogenic substances. Warren's studies showed that women afflicted with cystic disease before the menopause were 112 times as likely to develop cancer as those in the general population. He found that, after the menopause, this danger was not appreciably increased. On the other

hand, Bloodgood's school and other authorities, the weight of whose opinion cannot be overlooked, claim that the relationship between this condition and cancer is purely coincidental. Furthermore, our own follow-up studies on women having breast biopsies which showed advanced benign epithelial changes do not show any impressive increased incidence in malignancy.

Diagnosis.—Most cases of this disease are unquestionably subclinical, but an increasing number of women with cancer phobia are seeking medical counsel. Cystic disease is characterized by nodular or thickened areas in the breast varying in size which become increasingly painful before menstruation. The findings on examination are variable. There may be no cysts or the cysts may be so small as to be imperceptible or they may be as large as an egg and may be single or multiple. Large cysts just under the skin have a bluish translucency and have been termed by Bloodgood, blue-domed cysts. Most areas of nodularity consist of fibrous involution and are found in the upper outer quadrant. The pull of gravity due to the upright position has been suggested as a cause for this. Benign epitheliosis per se is not a palpable lesion and can only be diagnosed microscopically.

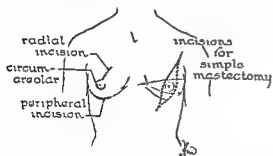


Fig 132—Common incisions used in breast surgery

Treatment.—Local excision of the diseased area is far from satisfactory. As the lesion is more diffuse than clinically apparent, the only cure is simple mastectomy. From the data presently available it seldom

appears that such a radical measure is justified. While the slightly increased incidence of cancer with cystic disease is generally recognized, clinical statistics do not support a radical approach just for the sake of the surgeon's and patient's "ease of mind." Before simple mastectomy is considered, the patient's marital status, age and familial history of cancer and personal feelings must be given due consideration. Naturally, a conservative attitude should be taken in the case of young, unmarried girls. However, any "dominant" lumps or suspicious areas must *always* be excised and microscopically examined. It may be argued that biopsy may miss an important area. There is no answer to this argument. If the biopsy shows advanced epithelial anaplasia, some surgeons unhesitatingly perform simple mastectomy. While it is an easy and simple matter for the surgeon to discharge his responsibility for preventing cancer by simple mastectomy, the patient's emotional happiness, operative risk, and other factors must be appreciated. On the other hand the clinical distinction between benign and early stage malignant lesions is treacherous, and experienced surgeons are constantly distressed by the number of inoperable cancer cases that have been treated conservatively for cystic disease until the real diagnosis has become obvious. Every breast lump *must* be excised, but the real difficulty lies in the differential diagnosis between a lump and an area of nodularity. Cystic disease has a remarkable tendency to disappear during the first three months of pregnancy.

BENIGN TUMORS OF THE BREAST

Nonindigenous benign tumors such as lipomas, chondromas, dermoids, and sebaceous cysts may occur.

1. **Fibroadenoma.**—This type of growth makes up about 15 % of all breast tumors. It is most often seen as a firm, round, mobile, encapsulated, painless mass, about $\frac{1}{2}$ to 2 cm in diameter in women under the age of

25 who have not yet nursed. Such tumors may be single or multiple and are often associated with either localized or generalized cystic disease. They seem to arise from an overgrowth of the intralobular fibrous connective tissue. Treatment is local excision of the tumor, preferably under general anesthesia. The incision should be made radially to the nipple or along the inferior fold of the breast. In rare cases, cancer can be traced to proliferation of the involved epithelial cells and occasionally sarcomatous degeneration of a fibroadenoma occurs.

2. *Duct papilloma* generally grows slowly within a cystic dilation of one of the main ducts near the nipple and is characterized by a bloody discharge in some cases. When single, these tumors are smooth, firm, and round. If the tumor can be accurately delineated (by transillumination and digital pressure producing nipple discharge over a specific area), local excision is satisfactory as they are not prone to malignant change.

TRAUMATIC FAT NECROSIS

Traumatic fat necrosis should always be considered in the diagnosis of a breast lump although it is uncommon. Clinically, the lump cannot be differentiated from carcinoma. Occasionally following a blow, the liberated fat from ruptured cells is saponified by circulating enzymes. The lipogranulomatous reaction characterized by proliferation of fibrous tissue results in a firm, hard, generally painless lump of indefinite outline. The treatment is local excision of the mass for diagnostic purposes.

GALACTOCELE

This rare lesion forms a smooth, tense, rounded swelling of the breast during the second or third month of lactation. It is the result of blockage of a milk duct by scarring or local inflammation. Its recognition is important insofar as it may be confused with malignant disease. The treatment is repeated aspiration and support of

the breast by a compression bandage. Occasionally it is wise to arrest secretion by *Stilbestrol*.

PLASMA CELL MASTITIS

Plasma cell mastitis is a rare lesion of the lactating breast. It represents a foreign body reaction to the products of breast secretion which have escaped into the breast structure from a duct. Biopsy is necessary to distinguish it from carcinoma.

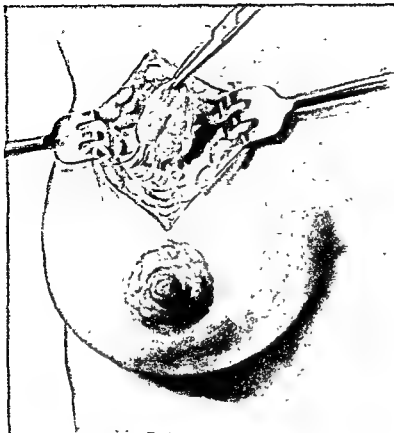
EXAMINATION OF THE BREASTS

The student should adopt a routine method of examining all breasts, and the following outline is suggested.

The patient is undraped to the umbilicus and seated on the edge of the table or bed with the arms at the sides. The lighting must be adequate and free from distorting shadows. The breasts are first critically inspected and compared for size, contour, and symmetry. The arms are raised and the examination is repeated. Next, the nipples are examined for eczema and retraction. The examiner now stands behind the patient and palpates for supraclavicular glands—a sign of secondary spread.

Then, with the pectoral muscles relaxed by placing the patient's arm on one of his own, the axilla is gently palpated in all directions with the fingertips. The location, size, consistency or fixity of any lymph gland is noted. The fact that a clinical search for involved glands is notoriously subject to error is no excuse for neglecting this procedure.

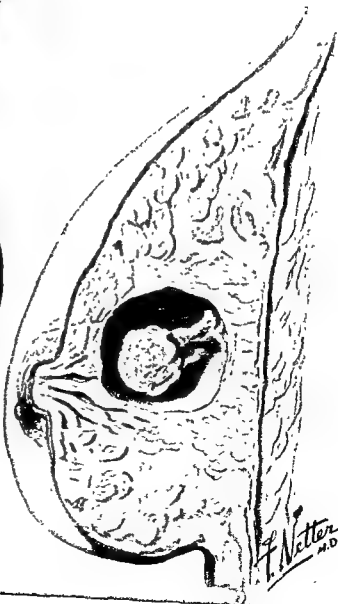
Palpation of breasts should always be done gently and kept to a minimum for fear of spreading cancer cells. Only the flexor side of the extended fingertips should be used. The breasts are palpated with the patient sitting up and then again with the patient in the supine position with the side of the chest raised with a pillow. The whole of the breast structure is then gone over gently with



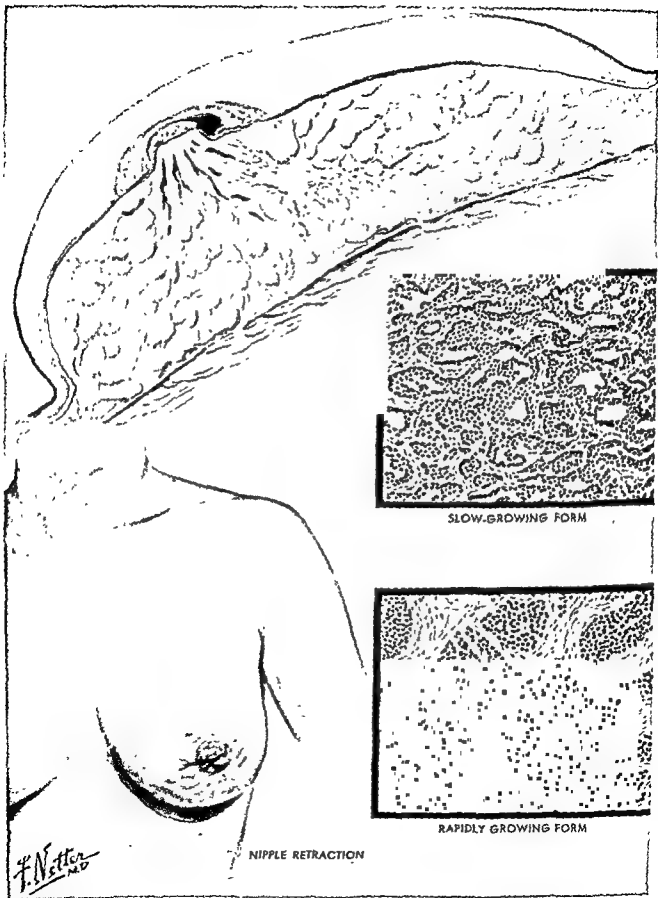
FIBRO-ADENOMA



BENIGN
INTRACYSTIC
PAPILLOMA



F. Netter
M.D.



SLOW-GROWING FORM

RAPIDLY GROWING FORM

NIPPLE RETRACTION

the extended fingers. The most difficult area to palpate with accuracy is directly beneath the nipple.

The skin of the breasts is now examined for areas of retraction or fixation. Edema of the skin or "peau d'orange" from lymphatic blockage is a late sign and usually denotes an inoperable lesion. By gently pinching and lifting the skin, retraction or fixation of areas is often elicited—a most important sign in cancer. Retraction is also elicited in other ways such as raising arms above the head, pushing the whole breast in each direction, compressing the breasts from both sides, contracting the pectoral muscles by pushing on the iliac crest with the hand or flexing the body on the hips with the neck extended.

Attention is again directed to the nipple to test for the presence of discharge. A milky discharge in a nonlactating woman has more serious significance than a clear watery or even bloody one. Bloody discharges are oftener found with benign lesions than with malignant ones. In fact only a very small proportion of breast cancers exhibit this sign.

Local x-ray examinations are of no help in examining breasts. Cytological examination following aspiration biopsy of lumps has proved unsatisfactory and theoretically the procedure may help to disseminate tumor cells. Aspiration of cysts may relieve discomfort from increased tension, but it is not a recommended procedure because of the danger of overlooking malignant disease. It may be justified in young women when only one cyst is present. The risk of disseminating cancer cells is probably less if the whole tumor is excised together with an adequate amount of surrounding breast substance than if the tumor is deliberately incised for frozen section purposes. This point, however, is still subject to debate.

In describing a lump in the breast, the following points should be noted: the size, shape, position, consistency, mobility, over-

lying skin dimpling, skin edema, nipple retraction, and nipple discharge.

Every patient should receive a thorough radiological examination of the chest, spine, and pelvis before radical mastectomy is contemplated, so that gross distant metastasis may, in some measure, be ruled out.

CANCER

Breast cancer, a disease of involution, is one of the commonest forms of malignancy in females. It is uncommon in males.

While striking examples of familial incidence are seen, there is no definite proof that the tendency is inherited. Little is known as to its etiology, although its higher incidence in nulliparous women and in those who have had some abnormality in lactation suggests a possible hormonal factor. Trauma, often related to the discovery of malignant disease, has not yet been incriminated as a definite etiological fact. Lawsuits are sometimes dependent on this supposition.

Pathology.—The many diverse pathological forms make precise classification difficult but a relatively simple one is suggested.

1. **Scirrhus Carcinoma** is the most common type of breast cancer. It forms a stony hard tumor in which the dispersed epithelial cells grow in solid nests and columns. The malignant cells appear to be compressed by the greatly increased fibrous tissue growth so that there is no attempt at glandular formation.

2. **Medullary Carcinoma** has a marked glandular proliferation with relatively little connective tissue. The lesion is soft and friable and microscopically consists of masses of large, round, epithelial cells.

3. **Adenocarcinoma** may be divided into the *mucoid type* with a tendency to papilliferous formation and the laying down of mucoid material, and the *ductal type*. The latter arises from the ducts of the breast parenchyma. Adenocarcinoma, while a relatively uncommon form of breast cancer, of-

fers a somewhat better prognosis than does the scirrhous carcinoma

4 **Intraductal Carcinoma (Comedo Cancer).** At first malignant cells remain confined to the ducts, and in this state the differential diagnosis between actual malignancy and secondary epithelial hyperplasia may be impossible. However, eventually the cells break through and invade the surrounding tissue.

Paget's Disease of the Nipple

Page's disease of the nipple is a chronic, intractable eczematoid change in the skin and occurs when a carcinoma, either of the underlying breast tissue, or of the ducts close to the nipple, has invaded and locally blocked the superficial lymphatic return. Cancer cells, which are here termed Paget's cells, invade the epidermis and become swollen and vacuolated. It is doubtful that the malignancy in these cases ever actually arises in the overlying squamous epithelium, but this point is still subject to argument. However, the disease is from the outset a malignancy, and the treatment is mastectomy. The important point is that an area of eczema in the region of the nipple lasting three weeks or more should be biopsied. Similar lesions may occur elsewhere in the skin of the body.

Breast Cancer—Clinical Features

In the early stages it may be impossible to distinguish carcinoma from other breast swellings. Thus, every lump in the breast of a woman must be considered as cancer until it has been removed and the proper diagnosis has been made by histological examination. Nodularity developing in one section of an otherwise normal breast may mask an early malignant change. A typical breast cancer is hard and is best felt by the flat of the hand compressing the organ against the chest wall. The edge of the tumor is vague and indefinite. With extension of the disease, there is ultimately skin involvement and the development of peau

d'orange. If the tumor is in the subareolar area, the nipple becomes retracted. Later the mass becomes fixed to the skin and to the pectoral fascia and muscles. If the tumor lies within, or has invaded one of the main ducts, there may be a nipple discharge which is usually clear but may be bloody. In the more diffusely growing forms, the tumor may involve the whole breast.

To assist in standardizing treatment and assessing results, four principal stages of breast cancer have been defined by Portmann.

Stage 1:

Tumor—localized in breast and mobile

Skin—not involved.

Metastases—none.

The treatment is radical mastectomy followed by deep x-ray therapy. After such treatment, five year survival may be expected in 70 to 80 % of cases and about 40 % will survive ten years.

Stage 2:

Tumor—localized in breast and movable

Skin—not involved.

Metastases—a few axillary nodes, none elsewhere.

With treatment as in Stage 1—one may expect 25 to 30 % five year survivals and about 12 % ten year survivals.

Stage 3:

Tumor—diffusely infiltrating breast, fixation to chest wall, edema of breast and/or secondary tumors.

Skin—edematous, indurated, ulcerated and/or secondary nodules.

Metastases—many axillary nodes involved or fixed but no clinical or x-ray evidence of remote metastases.

While the outlook in this stage is hopeless, worth-while palliation is sometimes achieved by mastectomy, but patients in this category are usually treated with x-ray and hormones.

Stage 4:

Tumor—as in any other stage.

Skin—as in any other stage.

Metastases—axillary and supraclavicular nodes extensively involved, clinical and x-ray evidence distant metastases.

X-ray or hormone therapy occasionally affords temporary palliation.

In assessing end results and to determine the value of treatment, it is necessary to compare the statistics with untreated cases. Daland in 1927 reported one hundred consecutive untreated cases and found that 40 lived more than three years, 22 lived more than 5 years, 9 more than 7 years, and 5 more than 10 years. Good results are sometimes obtained by the treatment of late cases and poor results from the treatment of so-called early cases.

Recent investigations have established the fact that by far the most important factor determining the outlook in breast cancer is the degree of malignancy of the original cancer cell (at a time when the tumor is not clinically apparent). In other words, the subsequent behavior of the tumor depends mainly on the growth characteristics inherent in the original parent cancer cell

Spread of Cancer

Cancer spreads by infiltration, lymphatic permeation, lymphatic emboli, by the blood stream and transcelomically. Infiltration peripherally from the primary source is the earliest type of spread. Columns of cells can be seen microscopically, transversing the tissue spaces and invading the surrounding structures. This method of spread is relatively slow. The commonest and most important method of spread is through the lymphatics by emboli to the regional glands. As a rule, the tumor cells pass deeply from the breast to the plexus of lymphatics lying on the pectoral fascia and thence peripherally. The most important route is to the axillary glands and secondarily, to the supraclavicular, infraclavicular, and cervical groups. The pleural cavity and mediastinum may be invaded by cells passing deeply to the glands along the internal mammary and intercostal vessels. The question as to

whether or not the lymphatics of the other breast form a primary target for lymphatic spread has not yet been settled. Involvement of the glands of the opposite axilla may occur. Some lymphatics pass downward through the rectus abdominis muscle and via the ligamentum teres to the liver. It is quite common for tumor cells in either the pleural or abdominal cavity to break loose and grow anywhere on the serosal surface. Lymphatic spread may cause a deposition of cells in the skin, particularly in the area of the operation wound. Cells from the thorax may pass to abdominal glands by the lymphatics of the posterior part of the diaphragm. This probably accounts for some of the metastases to the lumbar vertebrae, kidneys and adrenals. Spread by the blood stream occurs in late cases. Many cells no doubt enter the blood stream by direct extension while others pass into the subclavian vein via the thoracic duct, and hence to the bones and lungs.

Treatment

The treatment of breast cancer is radical mastectomy in all cases unless definite indications of inoperability exist. Contraindications to radical surgery include:

1. *Acute* (inflammatory) carcinoma and carcinoma developing in pregnancy.
2. *Local* advanced disease with widespread edema of the skin over the breast or carcinomatous nodules in the skin.
3. *Distant metastases*, i.e., involvement of the supraclavicular glands, bones, lungs, liver, etc. Palliative operations to remove or prevent skin ulceration may be indicated even in advanced cases.

X-ray Therapy

X-ray therapy does not cure cancer of the breast. However, tumor growth appears sometimes to be temporarily arrested and the life of the patient slightly prolonged when properly administered radiation therapy is given. There is great variation in the radiosensitivity of breast tumors. The best re-

sponse is from the more anaplastic types despite the fact that they carry the worst prognosis. Exposure to radiation results in local arteritis, thrombosis, atrophy, and necrosis of tumor cells. The resulting fibroblastic reaction may wall off some viable tumor cells. Objections have been voiced about the inadequacy of x-ray therapy and about the unpleasantness of radiation sickness, local skin reactions, and pulmonary fibrosis. However, we believe its benefits should be secured wherever possible in the treatment of all types of breast cancer.

X-ray therapy when combined with surgery may be given either before or after operation. Preoperative irradiation is given with the idea of destroying malignant cells which may have spread from the primary tumor and also to decrease the risk of spread at operation. It has occasionally made useful surgery possible by reducing the growth of an otherwise inoperable lesion. However, valuable time may be lost and healing delayed. At this institution postoperative irradiation only is preferred with the hope of destruction of malignant cells which have been left in the operative wound and of those disseminated as a result of the operation. X-ray therapy is often effective in relieving the pain of bony metastases.

Hormone Therapy

While the products of the endocrine glands must play an important role in this disease, hormone therapy is as yet in the experimental stage. The effects on breast cancer of oophorectomy and suppression of ovarian function by irradiation have been extensively investigated and the results do not justify this means of treatment. However, some strikingly palliative, although temporary, effects have been found attributable to the administration of various estrogenic substances and also to testosterone propionate. Although some effects of these hormones are diametrically opposed, there are certain similarities in action. They are

both stimulants in the sense that they improve muscle tone, increase hemoglobin, weight, appetite, metabolic rate, and invoke a sense of well-being. They may, however, give rise to edema as a result of salt retention. Hypercalcemia from testosterone has occasionally been observed. The evidence that tumor growth is sometimes definitely influenced is seen by the alleviation of the marked pain from bony metastases, the disappearance of carcinomatous nodules in the skin, the decrease in size of metastatic masses in the liver, and diminution in the amount of pleural fluid. Occasionally, on the other hand, there is a distinct acceleration of tumor growth following administration of either of these hormones. There is no indication for hormone therapy until the possibilities of surgery and x-ray treatment (which are much more effective) have been thoroughly exhausted.

Androgens

Testosterone is given in doses of 50 mg. intramuscularly three times a week for pain due to bony metastases which is intractable to x-ray therapy. It is also administered to women under the age of 60 with soft tissue growths nonresponsive to radiation therapy. It produces acne, coarsening of the skin, hirsutism, deepening of the voice, and stimulation of libido from increase in size of the clitoris. For maximum effect, a total dosage of about 3 grams is recommended and at least two months of treatment.

Estrogens

Estrogens are generally given to women over 60 or at least 5 years past the menopause with widespread involvement of soft tissues where irradiation is of no value. Diethylstilbestrol, 15 mg. daily for five months with a total dose of at least 2 grams, is recommended. This substance produces temporary nausea in some patients at the commencement of treatment and uterine bleeding in at least one third. It is con-

traindicated in younger age groups because it produces hyperplasia of mammary epithelium, enlargement of the breasts, and the changes associated with cystic disease. Carcinoma of the male breast apparently has resulted from estrogen therapy for carcinoma of the prostate. However, with the aforementioned indications, temporary subjective improvement can be expected in 60 % and objective benefit in 25 % of cases of female breast carcinoma.

Squamous Cell Carcinoma

Squamous cell carcinoma occurs as a relatively slowly progressing ulcerating lesion which takes origin from the overlying skin. Such tumors are inclined to be relatively indolent and they metastasize late. Treatment is either simple or radical mastectomy, depending upon the state of the growth.

Sarcoma

Fibrous connective tissue proliferation may be extreme and develop adenoid sarcoma, or the connective tissue cells may assume pure sarcomatous proliferation without glandular elements, taking the form of a spindle cell sarcoma. The prognosis in adenoid sarcoma, though the tumor is large and rapidly growing, is good. Even in spindle cell sarcoma, metastasis may take place only after the primary growth has reached a large size. The treatment is simple mastectomy if the tumor can be adequately encircled.

Radical Mastectomy

The classical operation was first described by Halsted. The patient is anesthetized, supine, and draped with the appropriate arm abducted to 90°. The mass is totally excised and examined microscopically to confirm the diagnosis. If the diagnosis is confirmed, the instruments, drapes, and gloves are changed to lessen the danger of dissemination of tumor cells. A blood transfusion is started. The incision begins over the insertion of the pectoralis major muscle

on the humerus. It curves upward at first—then inward and downward over the anterior chest wall on the lateral aspect of the breast staying at least 2" from the edge of the tumor. The other incision leaves the first point and passes medially to encircle the tumor. It then meets the first incision below the breast. The medial incision is then carried down to the pectoral fascia so as to excise all the breast tissue. The skin flaps must be kept as thin as is consistent with adequate circulation.

The division of the clavicular and pectoral part of the pectoralis major muscle is found and separated with the scalpel handle and followed down to the humerus. The head of the pectoral part is then detached at the humeral insertion, and the muscle is reflected downward exposing the pectoralis minor. The insertion of the latter is detached from the coracoid process and removed together with the surrounding clavipectoral fascia. The axilla is now accessible and its content of lymphatic glands, fascia, fat is carefully and thoroughly excised. The axillary sheath must be opened as it contains many lymphatics. If possible the long thoracic nerve to the serratus anterior and the middle subscapular to the latissimus dorsi muscle are spared. Dissection is now carried down until both pectoral muscles are detached at their origins. The upper part of the fascia over the rectus abdominis is also removed. The mass of breast tissue and muscle is now discarded. A stab wound is made in the lateral skin flap for a long soft rubber drain. Difficulty may be encountered in skin closure because of the large amount of skin which must sometimes be removed. If closure is impossible without undue tension, a split skin graft should be taken from the thigh and sutured in place. A firm, well-padded dressing is now applied. The mortality resulting from this operation should not exceed 1 %.

Arm movements are begun the next day following a change of dressing. On the second or third day, according to the amount

of discharge, the drain should be removed, and by this time the patient is usually made ambulatory. Sutures are removed from the sixth to ninth day if the healing process has been normal.

A distressing complication of the operation of radical mastectomy is edema of the arm. This is caused by disruption of lymphatic pathways, low grade infection, thrombosis of the axillary vein, or vascular fibrosis from x-ray therapy. It is treated by elevation, massage or the use of an elastic arm bandage.

breast cancer. This approach has met with much opposition on the grounds that it ignores the established principles of cancer surgery. While McWhirter subscribes to the dictum "if you cut out cancer, you cure it," the poor results from radical mastectomy where the axillary glands are involved have led him to investigate other methods of treatment. He states that in 40% of cases suitable for radical mastectomy the axillary glands are not involved and therefore their removal is unnecessary. When the axilla is invaded, the supraclavicular glands are in-

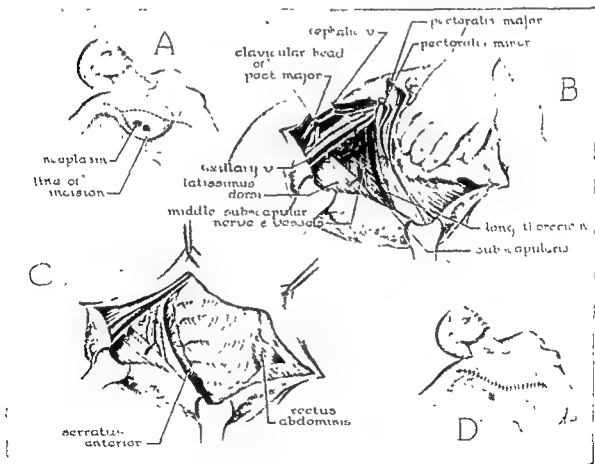


Fig 133—Radical mastectomy

Simple Mastectomy

Recently, a new school led by R. J. McWhirter, an Edinburgh radiotherapist, has been advocating simple mastectomy followed by x-ray therapy as a general treatment for

involved in 33% and the internal mammary glands in 48%. Furthermore extension of the scope of the radical operation as practiced by various surgeons so far has not shown any improvement in end results.

McWhirter firmly believes that axillary dissection in the presence of carcinoma serves to disseminate malignant cells (in a manner similar to bacterial spread) with shortening of the patient's life. He claims better results in his series since 1941 than other authorities with the present-day orthodox approach.

It is unlikely that any significant advance in the management of breast cancer will occur until such time as more fundamental information pertaining to the specific nature of the neoplasm is available. The student should be cautioned about the part played by statistical gymnastics and the careful selection of cases where any optimism is shown in articles dealing with breast cancer.

REFERENCES

- Boyd, W.: *Surgical Pathology*, ed. 6, Philadelphia, 1947, W. H. Saunders Company.
- Geschickter, C. F.: *Diseases of the Breast; Diagnosis, Pathology and Treatment*, ed 2, Philadelphia, 1945, J. B. Lippincott Company.
- Gordon-Taylor, Sir Gordon, McWhirter, R., and Cade, S.: Discussion; the Treatment of Cancer of the Breast, *Proc. Roy. Soc. Med.* 41: 118-132, 1948.
- Haagensen, C. D.: *Carcinoma of the Breast*, J. A. M. A. 138: 195-279, 1948.
- MacDonald, I.: *Biological Predetermination in Human Cancer*, *Surg, Gynec & Obst.* 92: 443-452, 1951.
- McWhirter, R. J.: Personal Communication, March, 1952.
- Park, W. W., and Lees, J. C.: The Absolute Curability of Cancer of the Breast, *Surg, Gynec. & Obst* 93: 129-152, 1951.
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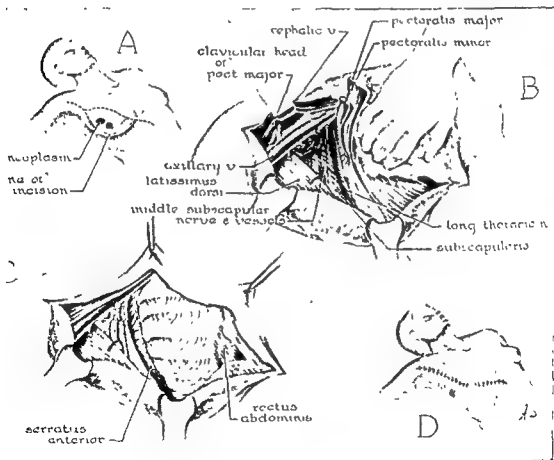


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REFERENCES

- Boyd, W.: *Surgical Pathology*, ed. 6, Philadelphia, 1917, W. B. Saunders Company.
- Geschickter, C. F.: *Diseases of the Breast; Diagnosis, Pathology and Treatment*, ed. 2, Philadelphia, 1915, J. B. Lippincott Company.
- Gordon-Taylor, Sir Gordon, McWhirter, R., and Cade, S.: Discussion; the Treatment of Cancer of the Breast, *Proc. Roy. Soc. Med.* 41: 118-132, 1948.
- Haagensen, C. D.: Carcinoma of the Breast, *J. A. M. A.* 138: 195-279, 1948.
- MacDonald, I.: Biological Predetermination in Human Cancer, *Surg., Gynec. & Obst.* 92: 443-452, 1951.
- McWhirter, R. J.: Personal Communication, March, 1952.
- Park, W. W., and Lees, J. C.: The Absolute Curability of Cancer of the Breast, *Surg., Gynec. & Obst.* 93: 129-152, 1951.
- Saner, F. D.: *The Breast*, Bristol, 1950, John Wright & Sons, Ltd.

CHAPTER XIV

THORACIC SURGERY

STEWART BAXTER, M.D.

Introduction.—The field of thoracic surgery has made tremendous advances during the past twenty-five years, and the volume of literature concerning this specialty has reached enormous proportions.

The early history of thoracic surgery is extremely interesting and was concerned chiefly with the surgery of tuberculosis. It has been well reviewed by Alexander. Modern thoracic surgery was given considerable stimulus by the discoveries of the antibiotic drugs, especially penicillin, streptomycin, and para-amino salicylic acid. These drugs have rendered existing procedures much safer and have greatly extended the scope of radical surgery for tuberculosis.

Applied Anatomy and Physiology.—The thoracic wall is composed of ribs and intercostal muscles attached to the spinal column, and moves under a combination of voluntary and involuntary control during respiration, coughing, and sneezing. It is important to remember that the thorax and abdomen overlap each other to a considerable extent, and that disease or injury in one may produce symptoms in the other, e.g., abdominal pain in basal pleurisy and pneumonia.

Physiology of Respiration.—Respiration is under voluntary (cortical) and involuntary (reflex) control. Its regulation is carried out by the integrated action of a muscular, nervous, and chemical mechanism under the control of the respiratory center situated in the medulla.

Muscular Mechanism.—The enlargement of the thorax during inspiration creates an inequality of pressure between the outside and alveolar air, and as the chest expands, air flows into the lungs. Inspiration is an active mechanism; expiration, essentially

passive. The intrapleural pressure is normally subatmospheric except during coughing and straining.

Nervous Mechanism.—Reflex impulses reach the respiratory center from the lungs, carotid body and sinus, and the aortic arch. Additional impulses are carried by the trigeminal, glossopharyngeal, and intercostal nerves. Impulses from stretched receptors in the lung, which shorten inspiration and initiate expiration are conveyed to the respiratory center via the vagus. The receptors of the cardioaortic and carotid sinuses act as a result of both pressure and chemical changes in the blood. An increase in arterial blood pressure induces reflex inhibition of the respiratory center, while lowered pressure stimulates the center, causing hyperpnea.

Chemical Mechanism.—Carbon dioxide and, to a much lesser extent, lactic acid, by increasing the hydrogen ion concentration of the blood, are the chief chemical regulators of respiration. An increase in pH stimulates respiration by direct action on the center and also, reflexly, by stimulation of the nerve endings in the carotid sinus and aortic arch. Lack of oxygen, such as in high altitudes, also causes increased ventilation.

CONGENITAL ANOMALIES OF THE THORACIC CAGE

Certain somewhat rare congenital anomalies of the thoracic structures or contents require recognition, and occasionally operative correction.

Anomalies of Ribs

Complete absence of portions of one or more ribs, sternum, and pectoralis major muscles is occasionally noted. There is con-

siderable flattening and lack of function of that side of the chest. Anomalies connected with the first rib and cervical rib are discussed separately. Bifid ribs and fused ribs are seen not infrequently in routine films.

Hernia of Lung

Hernia of the lung may be congenital or acquired, due to trauma or infection involving some portion of the thorax. A true hernia exists when a portion of lung covered by parietal pleura protrudes through an abnormal opening in the thoracic wall, mediastinum or diaphragm. *Prolapse of lung* is a protrusion of lung through the parietal pleura.

Etiology.—Most of the cases are traumatic but rare congenital supraclavicular herniation through Sibson's fascia is encountered. Herniation may be due to large infected wounds of the chest.

Diagnosis.—The diagnosis is usually evident from the history and physical examination, there being a resilient, resonant tumor which appears on respiration, coughing and straining.

Treatment.—Small supraclavicular hernias may be completely asymptomatic and no operative treatment is indicated. Repair of Sibson's fascia can be performed in more severe cases. Repair of the traumatic types is more difficult, requiring frequently the use of fascia, periosteal flaps from adjacent ribs, and even the transplantation of ribs above and below the defect.

Pectus Excavatum ("Funnel Chest")

This is a rare congenital deformity of the chest due to depression of the costal cartilages inward with considerable backward displacement of the sternum, usually in its lower part.

TRAUMA OF THE THORAX

Trauma to the thorax may involve injury not only to the structures of the chest wall but also to its contents. Several traumatic mechanisms are recognized: e.g., direct and

indirect violence, blows and blast injury. There may be a break in continuity of the chest wall including the pleura, e.g., stab and gunshot wounds, or there may be tears and ruptures of the intrathoracic organs, lungs, heart, pericardium, esophagus, and great vessels. Consequently, for the sake of clarity, this subject will be considered under the following headings:

1. Nonpenetrating trauma of the thorax and viscera.
2. Penetrating wounds of the thorax.
3. Blast injuries of the thorax.

1. Nonpenetrating Trauma of the Thorax and Viscera

This may result from direct blows as well as crushing injuries. *Contusions and hematomas* of the chest wall rarely require surgical treatment. *Rupture of an intercostal artery* from stab wounds or rib fractures, not penetrating the pleura, may occasionally require open exploration and ligation.

FRACTURE OF RIBS

Etiology.—Direct blows, crushing injuries and, occasionally, muscular strain and severe coughing or sneezing may fracture the ribs. Pathological conditions such as cysts or tumors predispose to fracture. The most common sites are from the 4th to the 8th ribs along the anterior and posterior axillary lines and at the angles. These usually result from compression of the chest. The 1st rib may be fractured in downward trauma to the clavicular region. Types vary from simple, undisplaced, to grossly comminuted fractures which may be complicated by damage to the deeper structures.

Signs and Symptoms.—Pain at the site of injury and on respiration is characteristic. Marked voluntary splinting of the abdominal muscles on the affected side is usual. Localized tenderness and crepitus may be felt at the site of fracture. Tissue emphysema is only present if there has been rupture of the parietal pleura and lung, but the absence of this finding does not rule out lung damage. If the lung has been punctured, there may

be bloody expectoration. Careful x-ray study will confirm the diagnosis.

Treatment—The treatment of simple fractures involves some form of strapping, sedatives, and intercostal nerve block. *Strapping* can be done with adhesive tape, the conventional method of strapping the affected side to the unaffected side being most widely used. However, some find that complete circular strapping of the lower ribs is more effective regardless of the site of fracture. A well-fitted and applied chest binder has been found to be just as effective and does not require shaving of the chest. It can be readjusted and removed for x-ray and physical examination of the chest

organs is quite common. Reduction of the dislocation may be necessary, with fixation by strapping or by hyperextension plaster mold. Open reduction and fixation by suture or Kirschner wire are occasionally necessary if gross displacement is present.

"STOVE-IN-CHEST"

This refers to a severe crushing injury when one side of the thorax has been driven in. Numerous ribs are usually fractured, frequently in two places (e.g., the axillary line and angles), plus fracture of the sternum and other bony parts. Serious complications such as tension pneumothorax, mediastinal emphysema, injury to major vessels and me-

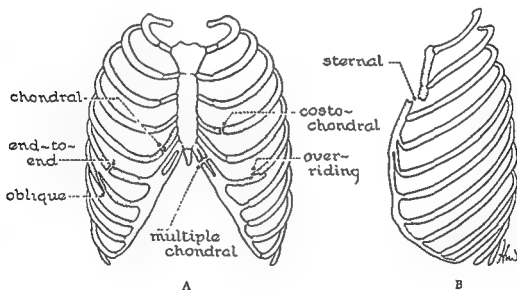


Fig 134—A. Types of rib and chondral fractures B. Fracture displacement of sternum

Regional nerve block is useful in relieving severe pain and respiratory embarrassment early in the condition. One or usually two intercostal nerves are infiltrated with 1 % Novocaine above and below the site of fracture or fractures, 3 to 5 c.c. being injected in each space.

FRACTURE AND DISLOCATION OF COSTAL CARTILAGES AND STERNUM

This is the result of either a direct blow or crushing injury. Injury to the intrathoracic

diastinal flutter, later, empyema, lung abscess and mediastinitis may result.

General supportive treatment such as oxygen, blood or plasma for shock, removal of bronchial secretions or blood is indicated.

Other complications will be discussed separately.

2. Penetrating Wounds of the Thorax

Penetrating wounds may be internal from comminuted fracture of ribs, or external from stab and gunshot wounds. Intrathor-

racic complications result from injury to the lung, heart, and great vessels. Among the conditions that may be encountered, the following are the most important and will be discussed individually:

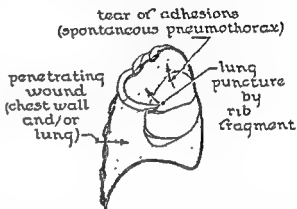
- (1) Collapse from massive hemorrhage.
- (2) Tension pneumothorax, with or without hemothorax.
- (3) Mediastinal emphysema.
- (4) Pneumo- and hemopneumopericardium
- (5) Laceration or rupture of diaphragm, with injury to the abdominal organs and immediate or subsequent herniation into the thorax.

Collapse from massive hemorrhage may be difficult to distinguish from primary traumatic shock. As a rule, collapse from shock is more rapid than that caused by hemorrhage, unless from some major source. In primary shock, the rapid weak pulse, clammy skin, and low blood pressure are present immediately following injury.

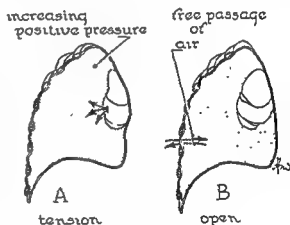
Tension pneumothorax and hemothorax result from any injury perforating the lung tissue when there is a nonadherent pleura. Comminuted rib fractures, with sharp spicules of bone driven into the lung, stab and gunshot wounds are the most common causes. Tension pneumothorax is the term used to describe air in the pleural cavity under increasing pressure due to a "ball valve" wound of the lung or bronchus, when air leaks out during inspiration, but cannot escape during expiration. In this way increasing positive pressure pneumothorax is built up, causing progressive collapse of the lung and mediastinal displacement. Blood or bloody fluid tends to collect in the pleural cavity adding to the compression.

Symptoms and Signs.—The symptoms and signs are usually diagnostic. There may be subcutaneous emphysema as an indication of lung damage, dyspnea, frothy bloody sputum, cyanosis, rapid weak pulse, lowering of the blood pressure, and hyperresonance with absent breath sounds over the affected

side. With accumulating blood or fluid there will be dullness at the base. There may also be evidence of mediastinal shift. All these symptoms and signs will become more acute if the condition is unrelieved, and the patient may go into general cardiorespiratory collapse and die.



Causes of pneumothorax (after Netter)



Types of pneumothorax

Fig 135—Causes and types of pneumothorax.

Diagnosis.—The diagnosis is made by the history, physical examination, confirmed by x-ray, if necessary; but in an acute stage this delay is not justified.

Treatment.—The treatment is to remove the air from the pleural cavity and restore mediastinal pressure relationships to normal. In an emergency, this can be simply achieved by inserting a large needle, No. 18 or 16 gauge, through an intercostal space, and the air will be heard to escape under pressure.

The needle is then attached to a pneumothorax apparatus, the pleural pressure measured and further air removed until pressures approach normal. Positive pressures of 15 to 20 cm. of water are often found.

If, after the removal of a significant amount of air (500 to 1000 c.c.), the pleural pressure rises again, a more continuous form of pleural decompression is indicated. An

aorta, vena cava, or hilar vessels cause such exsanguinating hemorrhage that the actual hemothorax is of secondary importance. However, hemothorax as an established condition requires separate consideration.

Pathology.—The presence of a fairly large amount of sterile unclotted blood in the pleural cavity may be relatively asymptomatic unless it compresses the lung, or causes

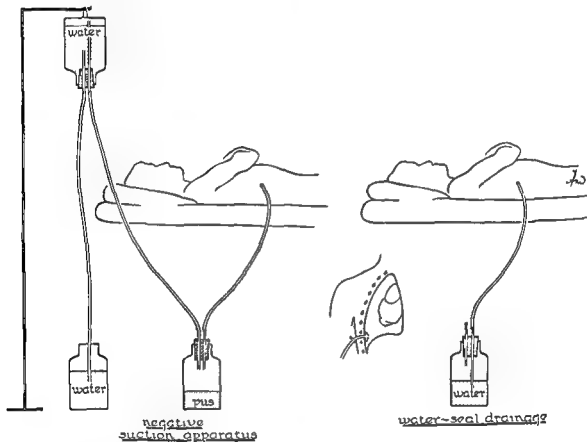


Fig 136 —Water seal and suction drainage

intercostal catheter attached to a water seal bottle or Wangenstein type of suction should be inserted. In this way positive pressure cannot recur, the opening in the lung will soon seal over and the lung will be encouraged to expand, at which time the catheter is removed.

Traumatic hemothorax may occur, with or without pneumothorax and from the same general causes as pneumothorax. Wounds of the major vessels such as the

enough mediastinal shift to produce respiratory or cardiac embarrassment. However, if allowed to remain in the pleural cavity, two complications may occur: organization and fibrothorax. These are caused by infection and clotting of the blood.

It has been well established that blood in the pleural cavity contaminated with bacteria or exudate from damaged tissues will clot, and soon layers of fibrin over the lung surface are formed. This leads to the con-

dition of organized fibrothorax, where the lung is encased in a tough fibrous membrane which seriously impairs its respiratory function.

Diagnosis of Hemothorax.—The diagnosis of small amounts of blood in the pleural cavity is difficult. Larger amounts give the characteristic signs of dullness and absent or distant breath sounds. X-ray shows the fluid level if a degree of pneumothorax is present as well.

Treatment.—Immediate aspiration of unclothed hemothorax is only indicated as an emergency measure to relieve respiratory distress, since increasing the negative pressure in the pleural cavity at this stage may only result in continued bleeding. However, in 24 to 48 hours, when conditions have likely become stabilized, aspiration can be started with safety, about 500 c.c. being removed daily or every other day until the pleural cavity is dry. Air should not be replaced, since the object is to keep the lung expanded. Penicillin, 200,000 to 500,000 units, in 10 to 15 c.c. of saline, should be instilled at the end of the first aspiration, and repeated if infection is suspected.

Clotted hemothorax may require rib resection and removal of clots, as in the open treatment of empyema. If the underlying lung does not show a tendency to expand after removal of clots, a catheter should be left in the wound, or placed through a separate stab wound, and connected to negative suction to encourage expansion of the lung. In the late case if the lung is encased in firm fibrous tissue and shows no ability to expand under positive pressure applied by the anesthetist, *decortication* should be undertaken. This is the peeling from the surface of the lung and visceral pleura of organized fibrous layers, thereby freeing the lung and permitting re-expansion. Fissures of the mediastinal and diaphragmatic surfaces should be freed as well.

Mediastinal emphysema may occur as a result of closed or open thoracic injuries, and is due to a tear in the mediastinal

pleura. Straining, coughing, and concussion may also cause it. It is diagnosed occasionally by the presence of crepitation felt above the sternal notch extending into the neck and face, and may be detected by x-ray. If sufficient pressure is produced, damming back of blood in the great veins occurs, and the patient expires from asphyxia.

The treatment is symptomatic and supportive unless localized pockets of air are demonstrated beneath the sternum, when aspiration often gives great relief.

Pneumo- and pneumohemopericardium are rare conditions which can result from lacerations of lung tissue close to the hilus, the air dissecting the fascial planes around the vessels and entering through a tear into the pericardial cavity. The heart shadow is enlarged by x-ray, and air may be demonstrated within the pericardium.

Laceration or rupture of diaphragm. Since the dome of the diaphragm rises to the level of the junction of the 4th or 5th rib with the sternum during expiration, the possibility of its being perforated during penetrating wounds of the chest must be considered when they occur below this level. Gunshot and shell fragments are responsible for many wounds of this type in warfare. The possibility of injury or perforation of abdominal organs must always be considered.

Pathology.—The possibilities of abdominal injury vary according to which side of the diaphragm has been penetrated. On the right side the liver covers its undersurface and prevents herniation of the intestines or other structures, and bleeding is usually not severe. On the left side, however, the stomach, colon, kidney, and spleen are all in close contact, and perforation of these organs usually gives rise to serious complications.

Diagnosis.—It is frequently quite difficult to determine whether penetration of the diaphragm and intra-abdominal organs has occurred in combination with wounds of the thorax. Abdominal splinting and rigidity may be present with thoracic injuries alone, or may take several hours to appear from

abdominal causes such as perforation of the colon. Perforation of the stomach is likely to cause early and acute splinting if the contents are spilled into the peritoneal cavity; also free air may be demonstrated by x-ray if perforation has occurred. However, in the presence of marked abdominal rigidity and rebound pain it is wiser to explore, since the mortality from well-conducted early exploration is considerably less than that accompanied by late exploration when extensive peritonitis has developed.

Injury to the spleen presents the usual left-sided symptoms, and kidney damage is accompanied by blood in the urine. When extensive laceration of the left side of the diaphragm is present, herniation of viscera may take place immediately or at a later date, and can be ascertained roentgenologically.

Treatment—Surgical exploration of both thoracic and abdominal regions is usually indicated. The decision to explore through the chest and diaphragm depends greatly on the individual case.

In the face of serious thoracic wounds with extensive hemothorax, the transthoracic approach will, of course, be indicated in order to restore maximum pulmonary function. Later, transdiaphragmatic exploration of the abdominal organs can then be performed. With minor chest symptoms, such as from stab wounds, the abdominal route is usually indicated. Right-sided thoracoabdominal wounds are better approached through the thorax due to the anatomical relationship.

The most convenient double exposure can be obtained by entering the thorax through the 8th interspace and continuing the incision on to the abdomen as far as necessary. The rectus muscle can be retracted medially or sectioned as required. An adequate exposure of all the upper abdominal organs can be obtained in this way.

Open Wounds of Thorax. Etiology—By open wounds, we mean those in which there exists an opening into the pleural cavity so large that the soft tissues cannot close it off,

and there is free exchange of air through the opening. For this reason they are often referred to as sucking wounds. War wounds, shrapnel and shell fragments, severe automobile and industrial accidents are the common causes. Most stab and bullet wounds are not true sucking wounds, as the tissues of the chest wall seal the opening.

Pathology.—With one side of the thorax open to atmospheric pressure, several deleterious events occur to upset the normal cardiorespiratory physiology, viz :

(a) *Collapse of lung on the affected side*. With an unclosed opening in the pleura, and if there are no adhesions to support the lung, the collapse is usually complete, i.e., 50 % of the respiratory capacity will be lost.

(b) *Mediastinal flutter*. When one lung is collapsed, due to atmospheric pressure in the pleural cavity, the inspiratory pull of the thorax will be unbalanced, and the mediastinum, if mobile, will be drawn across to the good side during inspiration and return to the normal position during expiration. This continual shift of the mediastinum is called *mediastinal flutter*, and is a large factor in the production of shock and respiratory embarrassment. It produces impaired function of the remaining lung and further tends to reduce the vital capacity.

(c) *Interference with cardiovascular function*. Atmospheric pressure in the pleural cavity also impedes the return of blood to the heart by pressure on the vena cava and hilar vessels. Also, shift of the mediastinum occurring suddenly causes a kinking of the great vessels which may further contribute to respiratory distress.

(d) *Stagnation of air in the bronchial tree*. Since the collapsed lung does not expand in unison with the good lung as in normal respiration, air tends to be sucked out of it during inspiration and forced back again during expiration. Thus there is a shift back and forward of a certain amount of stale air in the bronchial tree.

Treatment—Emergency treatment consists of immediate closure of the wound by tightly

applied moist pads, which in itself may produce marked improvement in the respiratory difficulty and general condition. Later care under proper conditions includes effective débridement of the wound under positive pressure anesthesia, control of bleeding vessels (intercostal), removal of foreign bodies or rib fragments, and repair of laceration of the lung. The chest wall is closed lightly, with negative suction drainage, after instilling penicillin and streptomycin into the pleural cavity. Tetanus antitoxin and polyvalent antigas bacillus antitoxin are given in accordance with routine surgical principles. The wound must be watched for evidence of infection in the muscle planes and reopened if necessary. Hemothorax, tension pneumothorax, pneumonitis, lung abscess and empyema must also be kept in mind as possible complications.

Some surgeons prefer to close the wound tightly and aspirate accumulated fluid and air, instilling penicillin and/or streptomycin with each aspiration. However, negative suction or water seal drainage removes the fluid continuously and tends to keep the lung expanded; this is a definite advantage.

3. Blast Injuries of the Thorax

A blast explosion produces a momentary marked increase in air pressure immediately followed by a negative or suction wave. Injury may be sustained during both phases, but it is generally agreed that the initial pressure wave is the more destructive. The same principle acts in immersion blast, affecting, of course, only the immersed parts. The effect of bomb blast varies with the size of the missile, and is most severe in the atomic bomb blast.

Pathology.—There is a sudden excessive increase in the intrapulmonary and mediastinal pressures. In the lungs, rupture of the vessels and alveoli fills the area with bloody exudate which seeps into the bronchi and is expectorated as a bloody, frothy sputum. The heart experiences a tamponading effect,

squeezing the blood out of the thin-walled auricles into the pulmonary veins and vena cava.

Signs and Symptoms.—There may be a latent period before signs of shock, dyspnea, cough and bloody expectoration begin. There may also be signs of an acute abdominal or cerebral condition. Atelectasis may be produced by retained secretions.

Treatment consists of complete bed rest, efforts to clear the bronchial tree, and support of shock. Fluid and morphine should be given sparingly. Intranasal oxygen or an oxygen tent is helpful. Occasionally intrabronchial suction will be indicated. Wounds or injury to other systems, of course, should not be overlooked, and appropriate treatment should be given.

Death occurs from hemorrhage, respiratory insufficiency, cerebral injury, cardiac failure, and air embolism.

ACUTE PYOGENIC INFECTIONS OF PLEURA, LUNG, AND MEDIASTINUM

Empyema is defined as pus in the pleural cavity.

Etiology.—The incidence of empyema has markedly decreased since the widespread use of antibiotics in the pneumonias. The most common etiological organisms are the pneumococcus, streptococcus, and staphylococcus. Other organisms found in mixed or putrid empyemas include Vincent's group, Friedlander's bacillus, *E. coli* and various anaerobic streptococci. The routes by which the pleural cavity may become infected are: (1) from disease of the underlying lung, e.g., pneumonia of all types, lung abscess, bronchiectasis, infarction of the lung; (2) secondary to mediastinal infection; (3) introduction from outside, e.g., stab wound, pneumothorax, etc; (4) extension from a subdiaphragmatic abscess, liver abscess or chest wall infection; (5) via bloodstream as part of a septicemia; (6) infection of hemothorax due to any cause.

Pathology.—The pathology produced by an empyema depends on the infecting organism, the amount of effusion produced, and the site of the effusion. Mixed empyemas are far more toxic than those due to a single organism.

Anatomically, an empyema may be diffuse, i.e., involve the whole pleural cavity, or encysted, \equiv g, interlobar, diaphragmatic, mediastinal, apical, etc.

Empyema necessitatis indicates the relief of an empyema by spontaneous perforation of the chest wall.

sounds and usually some tympany and egophony in the compressed lung above the pleural fluid.

Diagnosis.—The diagnosis of empyema, apart from the history and physical signs, is confirmed by aspiration and x-ray. Bacteriological examination of the fluid removed should always be made, as well as notation of the physical characteristics of consistency, color, and odor.

Treatment.—The treatment of empyema has three aims: (1) to relieve the respiratory distress; (2) to reduce toxicity; (3) to



Fig 137—Massive pneumococcal empyema treated by continuous suction drainage and later by rib resection and open drainage

A X-ray before drainage shows massive effusion with marked displacement of the mediastinum to the right

B Final result after drainage tube removed

The degree of respiratory embarrassment caused by an empyema depends largely on the amount of lung tissue collapsed by the fluid, and the degree of mediastinal shift.

Signs and Symptoms.—Empyema usually follows a previously known pneumonia and is diagnosed by continuing fever, dyspnea, dullness, absent breath sounds and voice

promote reexpansion of the collapsed lung and maintain it until adhesions have formed and obliterated the empyema cavity. These aims can be accomplished in a variety of ways, depending on the amount and type of fluid present. The various procedures are discussed under the heading of *open* and *closed* drainage.

Closed drainage may be continuous or intermittent. *Open drainage* refers to a thoracotomy and the placing of a drainage tube directly into the empyema at its lowest level, thus producing open dependent drainage. *Continuous drainage* is performed by introducing an intercostal catheter into the empyema cavity and attaching it to a Wangenstein type of suction apparatus. In this way the fluid may be removed completely under controlled pressure and speed, forcing the lung to reexpand to fill the empyema space.

treatment alone may abort or cure an early empyema. However, late empyemas do not respond well to this form of treatment, and late reinfection and reaccumulation of pus, multiple loculation and organizing fibrothorax are possible complications.

Chronic Empyema, Early and Late

Early chronic empyema may occur from two to six weeks after the effusion has become frankly purulent. It is characterized by organization of fibrin layers on the pleural

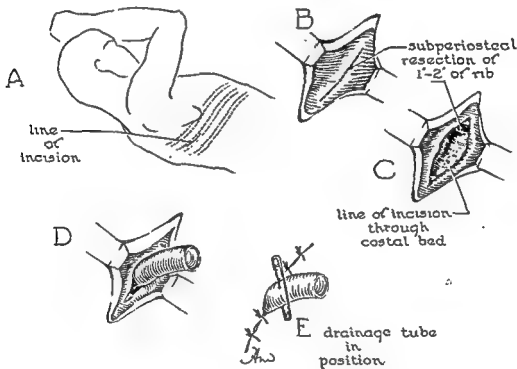


Fig 138 —Technique of rib resection and open drainage, for empyema.

In general, the open method is indicated in all thick, purulent empyemas with localized margins and stable mediastinum, while the closed type of drainage is of value in early, massive watery effusions without localization or fixation of the mediastinum.

Intermittent closed drainage is merely a periodic aspiration of fluid without air replacement, using a closed system. This procedure is usually combined with the instillation of a suitable antibiotic (penicillin, streptomycin) according to the bacterial flora. In a percentage of cases this form of

surfaces, and ordinary treatment in this stage may not bring about complete reexpansion of the lung. At this time, exploration of the thorax, decortication (or peeling off the fibrin layer from the visceral pleura) followed by continuous closed drainage will usually bring about complete reexpansion of the lung.

Late chronic empyema generally results from an improperly treated original empyema. Inadequate drainage, drainage too late in the disease, too early removal of tubes, retention of foreign bodies in the pleural

cavity (tubes, gauze, sponges, etc.) and bronchopleural fistula are common causes. In these cases the degree of fibrosis is so marked that decortication is often impossible or too hazardous, and some other means of obliterating the cavity must be attempted. Sauerization of the cavity by removal of overlying ribs and thickened parietal pleura, as in the Schede type of thoracoplasty, is frequently indicated. The use of flap muscle grafts has been advocated to fill deeper cavities.

Lung Abscess

A lung abscess is a localized suppurative focus situated in the lung parenchyma, whereas gangrene of the lung is a more acute process with massive necrosis.

Etiology—Acute lung abscess may be single or multiple and depends on a variety of etiologic factors.

- (1) Acute pulmonary infections account for a large percentage of all lung abscesses, e.g., all types of pneumonia.
- (2) Aspiration of septic material during operations on the mouth, nose and throat, also aspiration of foreign bodies by children, may produce obstruction, atelectasis and abscess.
- (3) Septic emboli in the bloodstream from other suppurative disease or septicemia, such as osteomyelitis or abdominal infection, may precipitate a lung abscess.
- (4) Lung abscess may be a complication of other diseases such as bronchiectasis or carcinoma of the lung.
- (5) Trauma to the lung occasionally results in abscess formation.

A wide bacterial flora is found in most lung abscesses and includes ordinary pyogenic organisms such as streptococci, staphylococci, pneumococci, Vincent's organisms, *H. influenzae*, colon bacilli and numerous anaerobic organisms.

Pathology—Usually two clinical types are recognized: the putrid and the nonputrid lung abscess. This classification really refers

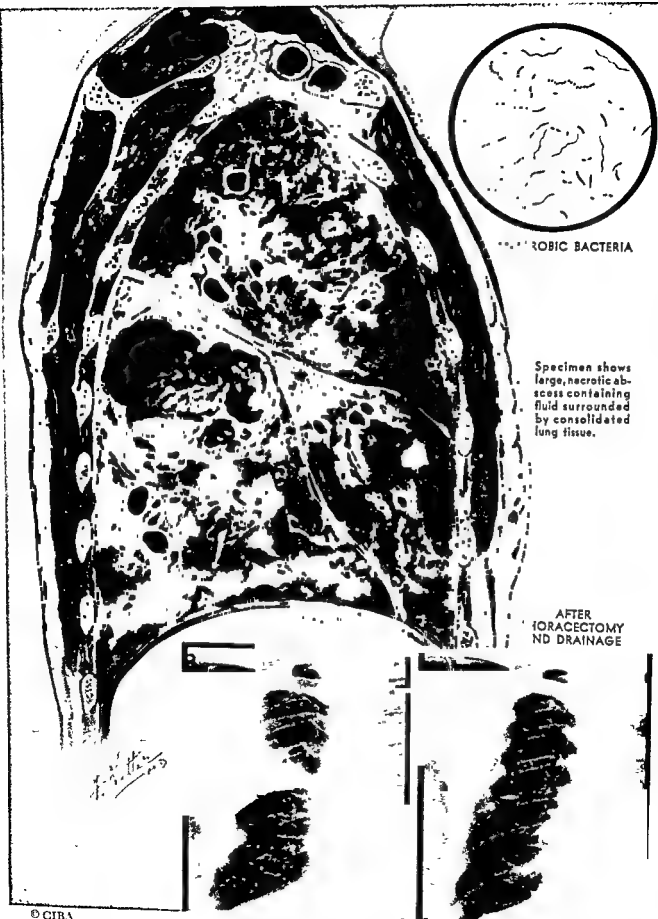
to the acuteness of the pathological process responsible for the formation of the abscess as a result of the etiologic factors mentioned above. The segment of lung involved becomes atelectatic and the alveoli are filled with serum, pus cells, and the parenchymal tissue is liquefied and destroyed. The blood vessels are thrombosed; this leads to further death of tissue, and a fan-shaped wedge of inflammation typically spreads out to involve the pleural surface and causes a pleuritis and obliteration of the pleural space over the area. Eventually the infective process may extend to involve the interlobar planes, mediastinal, or diaphragmatic surfaces.

During this phase there is a marked general reaction and the condition is termed *acute*. This stage lasts from four to six weeks. Cavitation forms at this time due to expectoration of necrotic lung tissue through a bronchus. Now the abscess tends to localize, a firm fibrous ring is formed around it, toxicity lessens, and the state is described as *chronic*. Death may occur from overwhelming toxemia, or perforation into a free pleural cavity may produce a lethal infection of an unprepared pleura. The chronic stage may persist for months or years unless properly treated, producing clubbing of fingers and toes, low-grade devitalizing toxemia and amyloid degeneration of the liver, spleen, and kidney.

Diagnosis—The diagnosis is made on the basis of the clinical history and physical signs. The latter, however, may be extremely slight, as in the case of a deep-seated abscess. The sputum is usually characteristic and copious—5 to 10 oz. daily.

Localization by x-ray is easy, especially when the cavity contains air and fluid. Bronchography is usually not of much value in the diagnosis of lung abscess since lipiodol rarely enters the cavity, but it may give an indication of the degree of bronchiectasis surrounding the cavity, especially in more chronic cases.

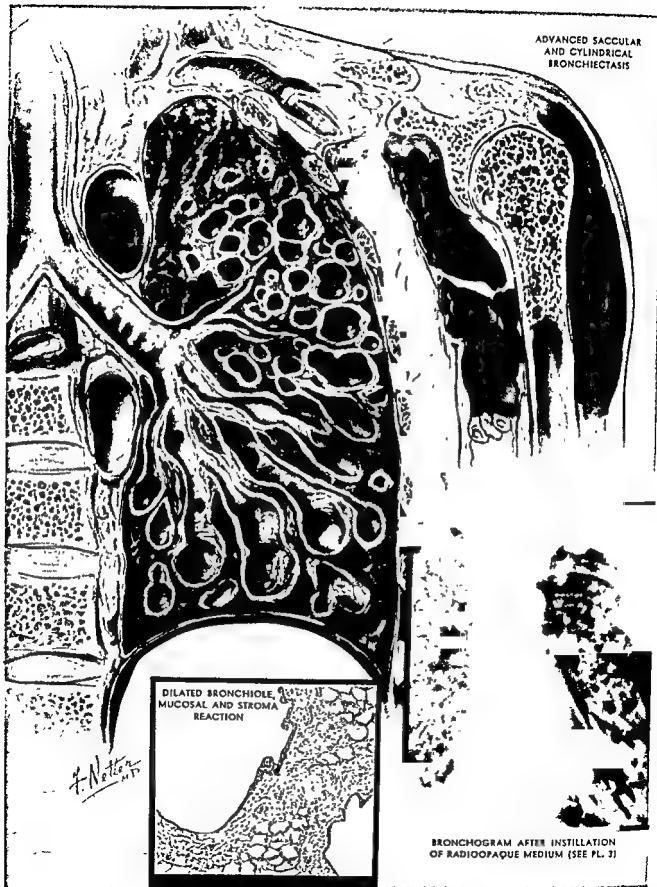
Treatment.—The antibiotics have greatly modified the clinical course of acute lung



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Plate IX.—Putrid Lung Abscess.

ADVANCED SACCULAR
AND CYLINDRICAL
BRONCHIECTASIS



DILATED BRONCHIOLE
MUCOSAL AND STROMA
REACTION

BRONCHOGRAM AFTER INSTILLATION
OF RADIOOPAQUE MEDIUM (SEE PL. 3)

abscess and have lessened to a considerable degree the number of cases requiring surgical treatment. Penicillin and/or streptomycin given intramuscularly, and by aerosol route as well, will frequently bring the temperature down to normal in a few days if started early in the disease. When a well-developed cavity is present, the results are not so dramatic, but nevertheless, with postural drainage, bronchoscopic aspiration and general supportive treatment, many abscesses will regress and heal, leaving little x-ray evidence behind. However, bronchography later on may reveal some residual bronchiectasis in the area.

Abscesses not responding to this form of treatment may require open operation and drainage after exact localization has been ascertained by x-rays in various diameters. The approach should always be through a sealed off pleural space, and if on exploration the pleura is found to be free, iodoform gauze is packed into the wound and the wound is closed tightly to allow pleural adhesions to form. About 10 to 14 days later the packing is removed and the abscess opened by diathermy cautery.

Chronic lung abscesses with loculation and bronchiectasis of the surrounding lung occasionally require lobectomy or pneumonectomy for a radical cure.

Bronchiectasis

Bronchiectasis is the term used to describe any permanent dilatation of the bronchial tree which is usually accompanied by infection.

Etiology.—This condition may be (1) congenital, or (2) acquired.

Congenital Type: In rare instances children may be born with a developmental deformity of the bronchial tree of one lobe or a whole lung, which later, becoming infected, gives the picture of advanced bronchiectasis at an early age. This latter condition is often referred to as honeycomb lung. Associated with this disease are various degrees and types of cyst formations of the lung.

Acquired Type: The factors responsible for acquired bronchiectasis are pneumonia of lobar or lobular type; whooping cough; bronchitis; pneumonitis from any cause, including tuberculosis, chronic sinusitis, and lung abscess. Chronic partial atelectasis arising from incomplete expansion at birth is also an occasional cause. Other lesions predisposing to obstruction, stasis and infection, are aspiration of foreign bodies, benign or malignant tumors of the bronchi. The symptoms may not develop to a sufficient degree for diagnosis until years after the initial predisposing cause.

Pathology.—The bronchi normally contain a certain amount of elastic muscular tissue which permits of a degree of contractility during forced respiration and coughing. As a result of chronic infection these tissues are replaced by fibrous tissue and the bronchi become progressively dilated from intrabronchial pressure during coughing. These dilatations are permanent and tend to increase. Thickening, scarring, and ulceration of the bronchial mucosa are frequently present. In longstanding bronchiectasis these bronchial dilatations may result in terminal abscesses containing purulent secretion, with surrounding peribronchial pneumonitis.

Several anatomical types of dilatations are described—cylindrical, fusiform, and saccular.

Anatomical Lobes and Segments of Lung.—A detailed knowledge of the lobes and bronchopulmonary segments of these lobes is of clinical importance to diagnosticians and surgeons who deal with bronchiectasis. Much recent work has been done to define these segments. Churchill and Belsey go so far as to state that the bronchopulmonary segment may replace the lobe as the surgical unit of the lung in bronchiectasis. Certainly it is becoming evident that segmental resection is indicated in some cases of bilateral bronchiectasis, when it is important to save as much normal lung as possible.

Each lung is considered to consist of four lobes, namely, upper, middle (lingular proc-

ess on left), lower and dorsal lobes, the dorsal lobe being the apical part of the lower lobe. These lobes, in turn, can be further separated into bronchopulmonary segments possessing a distinct bronchus, artery and vein with a relatively avascular plane between

in the second and third decades of life. There is often a history of repeated attacks of pneumonia or of longstanding sinusitis. The earliest symptom is morning cough and expectoration. This gradually progresses until the patient may bring up several ounces of

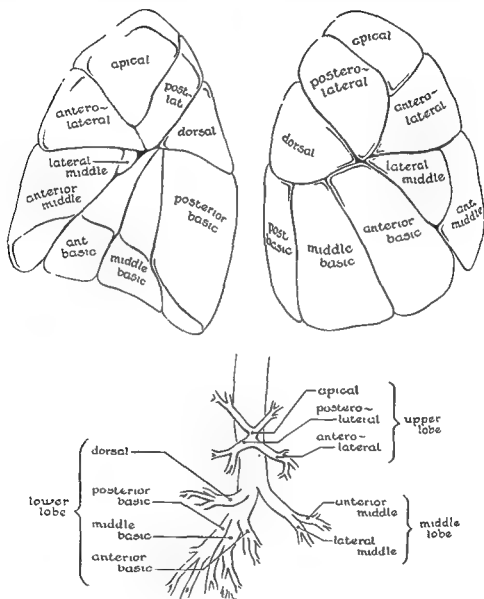


Fig 139—Lobes and bronchi of right lung

Signs and Symptoms of Bronchiectasis vary with the severity, extent and duration of the disease in each individual case. The initial pathological process frequently starts in childhood, and the symptoms appear only

increasingly fetid sputum during the day. Clubbing of the fingers develops and constitutional symptoms of malaise, cachexia, loss of weight, and pleural pain now appear. Hemoptysis may occur from time to time

and may be mistaken for the hemoptysis of tuberculosis. Life becomes a burden with incessant coughing of foul sputum. Fever, chills, and progressive emaciation lead, in the advanced untreated cases, to death during an acute flare-up. Amyloid degeneration is often seen.

of coarse, bubbling râles. Bronchography is necessary to outline the exact extent and clinical pattern of the condition. Bronchoscopy is usually performed. The blood picture is not characteristic, but reflects the toxic and nutritional changes. Tests for amyloid disease may show abnormal reten-

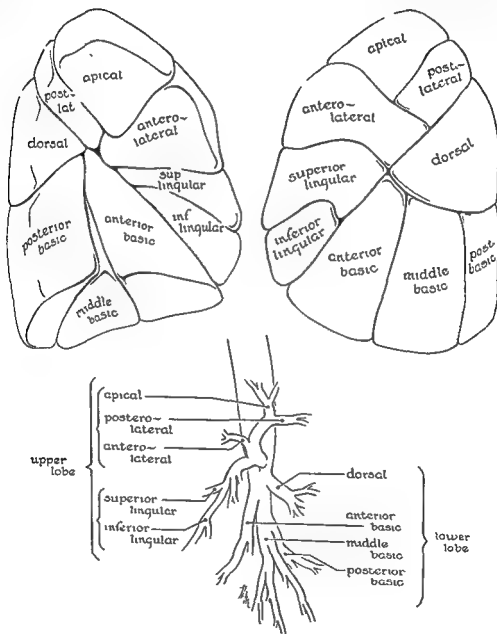


Fig 140—Lobes and bronchi of left lung.

Diagnosis.—The history including the diseases of childhood is important. The physical signs are those of impaired resonance at the bases, distant breath sounds and showers

of dye; the kidney function may be impaired.

Treatment.—In view of the underlying pathological process, it is obvious that in

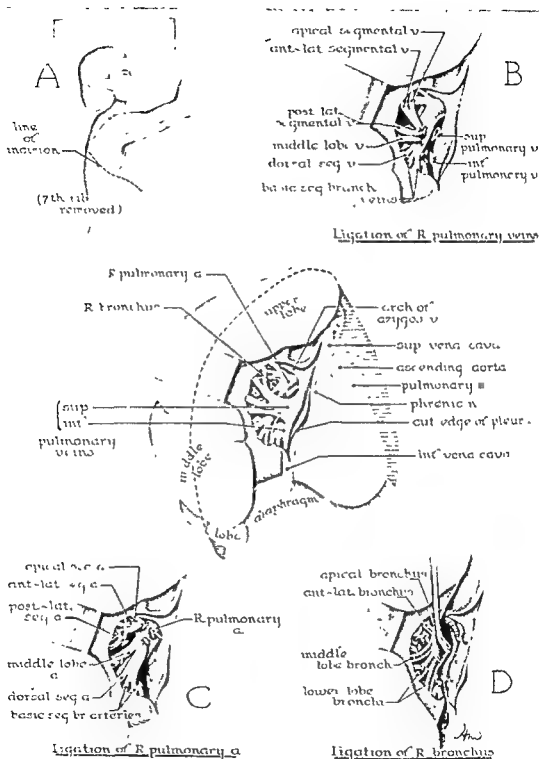


Fig 141—Hilar anatomy and technique of right pneumonectomy

well-established cases of bronchiectasis medical treatment is only palliative. However, it plays an important part in the preoperative preparation. Postural drainage, bronchoscopic aspiration, high-caloric and high-vitamin diet will improve the general condition. Autogenous vaccines are of value in certain cases. The antibiotics have been a great factor in the treatment of bronchiectasis. Aerosol penicillin and streptomycin will reduce the sputum in many cases and, carried on postoperatively, lessen the incidence of postoperative pneumonitis and empyema.

Lobectomy, segmental resection, and pneumonectomy, as indicated in the individual case, offer the only hope of a permanent cure. These operations, in capable hands, now carry a mortality rate as low as that in any other branch of major surgery. Many thoracic clinics are reporting consecutive series of 100 or more lobectomies without a fatality.

The modern technique of this operation is well standardized, the individual ligation of the pulmonary arteries, pulmonary veins, and bronchi being performed at the site of resection. Endotracheal anesthesia and high spinal anesthesia (Nupercaine) are most commonly used.

The posterolateral approach, resecting the 7th or 8th rib, is used for lower lobe lobectomy, and this or the anterior approach between the 3rd and 4th ribs in front for pneumonectomy. Negative suction drainage is generally employed for 24 to 48 hours postoperatively to remove the serosanguineous effusion and to encourage the remaining lobe or lobes to expand.

Complications.—The complications which may follow lobectomy are: (a) Early—hemorrhage from a major vessel; shock; atelectasis and postoperative pneumonitis, embolism, and pulmonary thrombosis. (b) Late—bronchopleural fistula, empyema, and wound infection. Rigid attention to operative technique and careful postoperative handling will eliminate many of the complications.

INTRATHORACIC CYSTS AND TUMORS

Cysts and Tumors of the Pleura

Cysts of the pleura are so rare that they have no practical significance.

Tumors of the pleura may arise from the secreting membrane itself (endotheliomas) or from the subserous layer. The latter are sarcomatous.

It may be difficult to differentiate tumors arising in the periphery of the lung from those of the pleura and chest wall. Pneumothorax may be of value. Bloody pleural effusion usually accompanies malignant tumors of the pleura. Tumor cells may be found on examination of the sediment. Metastatic involvement of the pleura is common.

Mediastinal Cysts and Tumors

Mediastinal cysts and tumors are frequently discovered by survey films and may be asymptomatic. Symptoms are usually caused by pressure on other mediastinal structures, viz, great vessels, esophagus, and nerves. The following classification modified from Bradford indicates the wide etiological range.

I. Congenital Cysts

1. Dermoid
2. Teratomas
3. Pericardial cysts
4. Bronchial cysts
5. Gastric and esophageal cysts
6. Cystic lymphangioma

II. Acquired Cysts

1. Parasitic
2. Neoplastic
3. Cystic hematoma.

III. Connective Tissue Tumors

1. Fibroma
2. Lipoma
3. Leiomyoma
4. Chondroma, chondromyoma
5. Sarcoma

IV *Neurogenic Tumors*

1. Neurofibroma
2. Ganglioneuroma
3. Neuroblastoma

V. *Thymic Tumors*

1. Benign thymoma
2. Malignant thymoma
3. Thymic cysts

VI *Primary Tumors of Lymph Nodes*

1. Lymphosarcoma
2. Hodgkin's
3. Sarcoidosis

VII. *Primary and secondary sarcoma*

VIII *Primary and secondary carcinoma*

IX. *Intrathoracic goiter*

X *Aneurysm*

The signs and symptoms of mediastinal cysts and tumors vary greatly with the etiology, and many are discovered accidentally during routine chest films. However, large tumors or cysts produce congestion of neck veins, substernal pain, dyspnea, dysphagia, cough, hoarseness and Horner's syndrome from pressure on the trachea, esophagus, great vessels, and nerves, respectively.

The diagnosis may be extremely difficult and only decided at thoracotomy and exploration. X-rays in various diameters, barium swallow, and pneumothorax assist in localizing the tumor. Fluoroscopy, kymography, bronchoscopy, and bronchography give added information in appropriate cases.

Venography and angiocardiology have been advocated recently as a distinct aid in diagnosis, particularly of aneurysms of the aorta or great vessels. Therapeutic x-ray therapy is useful in differentiating lymphosarcoma which shrinks considerably following this treatment from benign tumors and cysts which remain unchanged in size.

Treatment.—It is generally agreed that all mediastinal tumors and cysts should be removed surgically if there are no serious contraindications. Many of the benign tumors and cysts undergo malignant changes in later years. Perforation of an infected cyst into a

bronchus adds bronchiectasis or lung abscess to the existing condition and many require lobectomy or pneumonectomy as well.

Anterior mediastinal tumors are exposed by the anterior approach through the appropriate interspace, dividing the costal cartilage above and below. Occasionally, splitting of the sternum vertically and laterally into the 2nd or 3rd interspace is necessary to expose anterior mediastinal tumors such as thymomas.

Posterior mediastinal tumors are reached by the posterolateral approach.

Tumors and Cysts of the Lung

CYSTS OF THE LUNG

Cysts of the lung are relatively rare, but are being found with increasing frequency due to wider use of the x-ray. They may be congenital or acquired.

Congenital cysts. (1) Dermoid cysts and teratomas of the lung are reported, but are extremely rare. (2) Simple cysts containing air and lined by various types of epithelium are the most common. There is considerable controversy as to the origin of these cysts, but they may be solitary, bilateral or multiple. The multiple type may be associated with bronchiectasis or honeycomb lung.

Signs, Symptoms and Diagnosis.—Many cysts remain asymptomatic for years. Infection or rupture with spontaneous pneumothorax may cause the initial symptoms. With large multiple cysts, increasing respiratory distress is inevitable.

The diagnosis is made by the history and x-rays of the chest. Lipiodol will only occasionally enter a cyst since the communication with the bronchi may be extremely minute or tortuous.

Treatment.—Removal of the solitary cyst by dissection is feasible in some cases. The bronchial communications may be difficult to close. Lobectomy or segmental resection can be performed in certain cases.

Acute pneumothorax may be a recurring symptom and is treated by suction drainage

maintained for 2-3 weeks if necessary. A mild empyema if it results may cause adhesions and prevent subsequent ruptures.

Acquired cysts are believed to follow lung infection, pneumonia and pneumonitis, and are not to be confused with cavitation of lung abscess, bronchiectasis or cancer.

Emphysematous bullae or blebs are lung cysts without any epithelial lining and can present the same clinical picture as the true lung cyst.

the sequelae of atelectasis, pneumonitis, lung abscess and bronchiectasis.

Diagnosis.—Diagnosis of many of these tumors can be made by bronchoscopy and biopsy as they tend to originate in the large bronchi.

Treatment.—Local removal by the bronchoscope is only rarely advisable and lobectomy or pneumonectomy is generally performed, especially if infection of the lung has occurred distally.



Fig. 142.—Bronchial adenoma of left lower stem bronchus causing obstructive bronchiectasis and pneumonitis

BENIGN TUMORS OF THE LUNG

Histologically benign tumors are found in about 1% of the total cases of tumors of the bronchial tree. Fibroma, lipoma, adenoma, chondroma, hamartoma are some of the benign tumors occasionally reported. However, these tumors may undergo malignant change or cause bronchial obstruction with

Bronchiogenic Cancer

Etiology.—Cancer of the lung, once a relatively rare disease, has in recent years become one of the most frequent forms of malignancy encountered. It ranks third to breast and gastrointestinal cancers, and in the United States 150,000 to 200,000 individuals die of it yearly. It is more common

in males than females, 11 to 1, and may occur at almost any age, but is found most frequently in the 40 to 60 age group. Improved methods of diagnosis and a greater awareness of the public and medical practitioners for the disease account partially for its apparent increase, but heavy smoking, industrial gases, dust, etc., are regarded by some as etiological factors.

Signs and Symptoms.—Unfortunately, there are no characteristic early symptoms of cancer of the lung and even advanced cases are labelled "unresolved pneumonia," "bronchitis," "asthma," and with blood streaking of sputum, "tuberculosis." Consequently, in all cases presenting complaints referable to the chest, the possibility of carcinoma must be kept in mind.

The signs and symptoms vary with the size of the original tumor and the size and degree of obstruction of the bronchi involved.

Early symptoms include increased or unusual cough with mucoid, frothy and occasionally blood-streaked sputum. At this stage, there may be no actual obstruction, but as the tumor grows, wheezing cough or respiration may appear. Obstructive atelectasis and pneumonitis may lead to the "flu" syndrome of cough, fever, purulent sputum, and chest pain.

Late signs and symptoms include shortness of breath, pleural pain, loss of weight and strength which may be due to pleural effusion, empyema, carcinomatous abscess, and metastases to mediastinum, liver, brain, etc. Massive hemorrhages may occur. Involvement of phrenic and recurrent laryngeal nerves is occasionally seen.

Diagnosis.—The early diagnosis of bronchiogenic carcinoma offers the only hope of a permanent cure by pneumonectomy or occasionally lobectomy. Education of medical practitioners to assess and investigate adequately the nonspecific early chest symptoms of all individuals in the cancer age is important and is progressing as the full significance of the increasing incidence of lung cancer becomes generally apparent.

The diagnosis by history and physical examination alone is impossible except in advanced hopeless cases. Even x-ray diagnosis is difficult and uncertain in early cases. The main armamentarium in diagnosis is the use of the bronchoscope, bronchography, and cytological studies of the sputum or pleural fluid by competent specialists. Aspiration biopsies and even exploratory thoracotomy may be necessary before a positive diagnosis can be made and there should be no hesitation to do so in doubtful cases.

Pathology.—About 75 % of bronchiogenic carcinomas arise from one of the larger bronchi, while the remaining 25 % are more peripherally situated and some claim that they may arise from the alveoli (alveolar cancers).

The early changes include a thickening and roughening of the bronchial mucosa; later either a stenosis or blocking of the bronchus, penetration of the wall and infiltration of surrounding lung tissue occur. The tumor presents a greyish white appearance and larger masses may show hemorrhagic necrosis at their center. With obstruction of the bronchus, atelectasis, bronchiectasis, pneumonitis and abscess formation are common.

Large cancers may simulate lobar pneumonia while the more peripheral types may be difficult to distinguish from metastatic deposits or tuberculosis.

Extension occurs by direct invasion, lymphatic and blood stream metastases. According to Ochsner and de Bakey, as a result of investigation of 3,047 collected cases, the order of frequency of metastases is (1) regional lymph glands 72 %, (2) liver 33 %, (3) pleura 29 %, (4) lungs 23 %, (5) bone 21 %, (6) adrenals 19 %, (7) kidneys 17 %, (8) heart and pericardium 12 %.

Histology.—It is generally agreed that bronchiogenic carcinoma arises from the undifferentiated cells lining the basement layer of the bronchial mucosa. In spite of the cellular pleomorphism found in any one tu-



RADIOGRAM BEFORE AND
AFTER ATELECTASIS CAUSED
BY BRONCHOSTENOSIS

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Plate XI.—Advanced Bronchiogenic Carcinoma with Partial Obstruction of the
Right Main Stem Bronchus and Metastatic

mor, a predominating type of cell usually characterizes the histological structure of the tumor.

Three main types are recognized.

(1) An epidermoid or squamous cell type.

(2) Adenocarcinoma.

(3) An anaplastic undifferentiated type commonly referred to as "spindle" or "oat cell" carcinoma.

They occur in this order of frequency, and the squamous type is the least, and the anaplastic the most, malignant.

Treatment—The only treatment for bronchiogenic carcinoma is pneumonectomy and the removal of mediastinal lymph glands. Unfortunately, the number of cases in which this is possible is small and the five-year survival rate in the best centers is only 5 to 6 %. The use of x-ray or of nitrogen mustard is only palliative and does not change the ultimate outcome. The average survival of the inoperable case is about one year.

SURGERY OF PULMONARY TUBERCULOSIS

Historical and Introduction.—The surgical treatment of pulmonary tuberculosis occupies a prominent place in the management of this disease. Since its inception, about thirty years ago, many different operations have been devised to aid in the collapse and healing of diseased lungs. Some of these have not stood the test of time and have been discarded. The specific purpose of any surgical procedure in tuberculosis is to close and heal cavities and to render the patient bacillus-free.

The development of antibiotic drugs has improved the results of surgical treatment and may eventually become the treatment of choice. However, until such time as a drug is found that has selective action on the tubercle bacillus, surgery will be indicated in many cases.

Physiology and Pathology.—The physiological principle involved in any form of collapse therapy is that of selective rest of the

diseased lung. This is based on the premise that diseased lung tissue will heal more readily if its respiratory excursions are greatly diminished or abolished. Surgical therapy is not intended to supplant bed rest and general supportive measures; it is advocated as a means of curing cases when these measures have failed or do not give much promise of cure.

The most important effect of collapse of tuberculous foci is to increase fibrosis and to stimulate organization and encapsulation of diseased tissue. It increases perivascular and peribronchial fibrous tissue. Cavities are reduced in size until they are obliterated and healed. However, in spite of physically satisfactory collapse, healing does not always occur. Infiltration may be so firm and extensive that the lung is incapable of collapsing. Huge cavities may be reduced in size but fail to become obliterated. The term "cure" is usually not applied to tuberculosis in the true sense of the word, and encapsulated bacilli may become activated years after a clinical or apparent cure has been obtained. Thus the potential chronicity and insidiousness of pulmonary tuberculosis must always be kept in mind when evaluating the results of surgical collapse.

Artificial Pneumothorax (Intrapleural)

Artificial pneumothorax is the introduction of air into the pleural cavity under controlled pressures. While this form of collapse therapy is frequently undertaken by internists, its principles must be understood by the thoracic surgeon. Many of these cases come to some other form of surgery due to complications or inadequacy of the collapse, but nevertheless it is the most widely used single form of compression therapy.

Indications.—Since a satisfactory pneumothorax gives a greater degree of lung collapse than any other form of therapy, it should always be considered before any more radical or permanent form of collapse therapy, such as thoracoplasty. In general, the

only contraindications to pneumothorax are minimal or far-advanced disease. Thus early minimal cases should not be exposed to the few but real hazards of pneumothorax before a trial of sanatorium treatment, nor should hopelessly advanced bilateral cases be subjected to it.

Technique.—Air is introduced into the pleural cavity under controlled pressures using a standard pneumothorax apparatus. The initial injection is given over a normal area of the lung, usually in the midaxillary line or through the 6th or 7th interspace below the angle of the scapula. The area is carefully infiltrated with 1 % procaine down to the parietal pleura. A short bevelled or special initial pneumothorax needle (Kuss) is then inserted until the final resistance of puncturing the parietal pleura is felt. The needle is connected to the tubing from the pneumothorax apparatus and, if it is in a free pleural space, negative oscillations will be recorded varying from -12 to -4 cm of water on inspiration and expiration; 50 to 100 c.c. of air are now rapidly introduced in order to compress the underlying lung and prevent it from being punctured by the needle. Readings are again taken, and 400 to 500 c.c. of air are slowly introduced, the pressure being recorded several times. The final pressures are several cm. of water higher than the initial. If the pressures rapidly become positive after a few c.c. of air are introduced, the needle is either not in the pleural space or is in a pocket of adhesions, and further air should not be forced in at that site. If repeated attempts at various sites are unsuccessful, it must be concluded that pneumothorax is impossible to obtain. Refills of the successful pneumothorax are given at increasing intervals of from 2 to 14 days and checked by x-ray or fluoroscopy until the desired degree of collapse is obtained. An established case requires refills every 1 to 2 weeks; some only once a month, as the pleura becomes thickened and the rate of absorption from it becomes slower.

Artificial pneumothorax, if successful and uncomplicated, is maintained from 3 to 5 years in the average case.

The pneumothorax is abandoned gradually by increasing the interval between refills and by decreasing the amount of air given. The sputum should be checked repeatedly during this period and the pneumothorax re-established if bacilli appear in a previously negative sputum. X-rays are also taken to reveal reopening of cavities in the expanding lung. Some patients may experience discomfort on reexpansion of the lung due to change of mediastinal relations or traction on adhesions.

Complications.—

The complications of pneumothorax may be early and late.

Early pleural shock and air embolism may occur as a complication of the initial attempt. Pleural shock has been given as the explanation for the symptoms of unconsciousness, low blood pressure, and weak, rapid pulse that occasionally follow pleural puncture. Recovery usually follows ordinary supportive measures, but rare cases of death have been reported.

Air embolism may produce the same picture with the addition of twitching of muscles or limbs, and rarely permanent paresis and muscular weakness. It is due to puncture of the lung and the introduction of air into a pulmonary vein and, consequently into the systemic circulation, with the production of cerebral emboli and sudden death.

Pleural adhesions may complicate pneumothorax apart from causing difficulty at the initial injection. The lower part of the lung field may be quite free but the diseased area in the apex or upper lobe remains firmly adherent to the parietal pleura preventing adequate collapse. If the adhesions are present over a large area of lung and do not stretch with subsequent refills, the pneumothorax should be abandoned in favor of some other form of collapse therapy. Frequently, however, adhesions will stretch un-

til they become fibrous cords several centimeters long which are suitable for division by internal pneumonolysis.

Lung puncture without symptoms probably occurs fairly frequently during initial pneumothorax. Rarely hemoptysis, hemothorax or tension pneumothorax results.

Late complications are chiefly those of pleural effusion and tuberculous empyema. A very high percentage (65 %) of pneumothorax cases have small or moderate pleural effusions especially during the early months when adhesions are being stretched or torn. Serious effusions are usually due to tuberculous infection of the pleura.

Tuberculous empyema develops in about 10 % of all cases and constitutes the most serious complication of pneumothorax. Tubercle bacilli are generally found in purulent effusions. Secondary infection may occur resulting in a mixed tuberculous empyema. The prognosis in such cases is extremely grave.

Results of Pneumothorax Treatment.—The results of this form of treatment depend on several factors.

1. Character and extent of the lesion.
2. Degree of efficiency of the collapse.
3. State of the opposite lung.

In those patients with minimal lesions or a small cavity at one apex, when the lung is well collapsed, arrest of the disease may be expected much sooner than in a patient with extensive disease and thick-walled cavities. It has been estimated that at least 75 % of people, with unilateral disease and satisfactory collapse, control their disease if the pneumothorax has been maintained for at least 3 years.

Intrapleural Pneumonolysis

Intrapleural pneumonolysis refers to the intrathoracic division or enucleation of adhesions by means of the thoracoscope using the actual or diathermy cautery. This technique, first devised by Jacobaeus (1913), is being utilized with increasing frequency and

requires considerable experience and judgment on the part of the operator.

Indications.—The main indication is the presence of an incomplete pneumothorax with the diseased part of the lung attached to the parietal pleura by adhesions. Uncollapsed cavities, recurrent hemoptysis, and tuberculous laryngitis are conditions where optimum collapse is essential. The freeing of the adhesions does not always guarantee the success of the pneumothorax since positive pressure or valvular cavities may enlarge during pneumothorax. Bronchoscopy is frequently indicated prior to pneumonolysis to exclude the presence of tuberculous bronchitis or ulceration. The only absolute way of determining the presence of adhesions suitable for lysis is by exploratory thoracoscopy.

Technique.—Intrapleural pneumonolysis is one of the most difficult and potentially dangerous operations of intrapleural surgery. The instrument consists of an operating endoscope with direct or right-angled vision. The cauterizing electrode is introduced either through the same instrument or through a separate cannula. The operation is usually performed under local anesthesia with little distress to the patient.

The adhesions are treated in one of two ways.

1. Simple division by slow cautery or diathermy. This is suitable for all fairly long, relatively avascular adhesions.

- 2 Short thick adhesions can be enucleated from the parietal pleura, taking the base of their attachment with them. This is frequently a difficult and lengthy procedure. The following complications may occur.

1. Excessive hemorrhage from injury to the lung or other large vessel, i.e., subclavian or intercostal vessels.

2. Opening of lung tissue and/or cavity with tension pneumothorax, pyopneumothorax, tuberculous or mixed.

Results.—Results are judged by the completeness of the resulting pneumothorax, the

closure of cavities, and the absence of tubercle bacilli in the sputum. Only in those cases where a satisfactory division of all or most of the adhesions has been secured, can a successful outcome of the pneumothorax be expected. If pneumonolysis is unsuccessful or impossible, it is wiser to abandon the pneumothorax and consider other forms of treatment, i. e., thoracoplasty, phrenic nerve interruption, etc., according to the individual case

matic bags, etc., all of which have proved unsatisfactory because of irritating properties, or inability to maintain the degree of collapse required. Recently the use of Lucite balls (plastic) has been favorably reported as a compressing agent.

Indications.—The chief indication for extrapleural pneumonolysis is in cases where artificial pneumothorax is impossible, and thoracoplasty for some reason is contraindicated. The procedure has special applica-

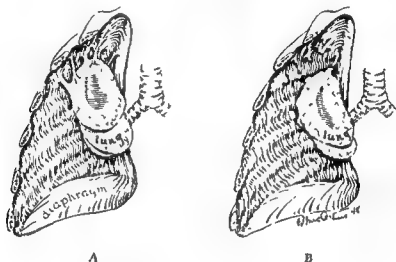


Fig 143 —A Artificial pneumothorax in a case with a large cavity in the upper lobe, which is attached to the chest wall by numerous adhesions
B Following division of adhesions, the lung is displaced toward the hilus with compression of the cavity

Extrapleural Pneumonolysis

(PNEUMOTHORAX PLOMBAGE)

Definition.—Extrapleural pneumonolysis is a procedure used to free the lung from the chest wall by stripping the parietal pleura from the endothoracic fascia on the inner surface of the thorax. In this way a large space can be formed which compresses the lung downward and medially. The separation can be carried down to the diaphragmatic surface if necessary. The resultant space is then filled with air (extrapleural pneumothorax) or with some nonirritating, noncollapsible material (plombage).

A variety of materials have been utilized in the past, paraffin, oil, muscle grafts, pneu-

tion in young children and women since there is not the same degree of deformity as results from multiple rib resection, i. e., thoracoplasty. Small apical lesions, with cavity and positive sputum, are ideal for this type of collapse.

Technique.—The extrapleural space is approached through a 2 to 3 inch resection of the 4th or 5th rib in the scapular region. Once the areolar tissue of the endothoracic fascia is reached, the stripping is rapidly completed by blunt dissection over the area to be collapsed. Care must be exercised at the apex not to injure the subclavian vessels. The opening in the chest wall is closed and the resultant cavity treated as an extra-

pleural pneumothorax. Alternatively, it may be packed with whatever material the surgeon favors before the chest wall is closed. Air refills have to be given every 24 to 48 hours for the first few days, the bloody effusion being removed at the same time. Much higher pressures are used than in intrapleural pneumothorax (+10 to +20 cm. of water), since this is a localized pocket, the maximum extent of which must be maintained to give adequate collapse.

Results.—A well-developed extrapleural space treated as a pneumothorax can usually be maintained for 12 to 18 months. The pocket tends to become obliterated by fibrous adhesions and scar tissue forming on the raw surface of the chest wall, which gradually draws the lung out. Occasionally the space may become infected with resulting pyogenic or tuberculous empyema. The complications of plombage are erosion into the lung and formation of a bronchopleural fistula, with expectoration of oil or wax when present. Migration of Lucite balls into the mediastinum or base of the neck has been reported.

Phrenic Nerve Interruption Operations

Definition.—Phrenic nerve interruption refers to the crushing, section or avulsion of the phrenic nerve in order to secure either a temporary or permanent paralysis of the diaphragm. Crushing or section as a rule gives temporary, while avulsion secures permanent, paralysis. Unilateral diaphragmatic paralysis causes the dome to rise 2 to 3 rib spaces, thus decreasing the volume of the pleural space and reducing the respiratory function and volume of the lung. On fluoroscopy, a "paradoxical" movement is noticed, the paralyzed diaphragm rising on inspiration and descending on expiration. The decreased activity of the diseased lung tends to promote healing and fibrosis even in upper lobe lesions. Basal lesions respond more favorably.

Indications.—Diaphragmatic paralysis is used chiefly as an adjunct to some other

forms of treatment or when pneumothorax is unobtainable and thoracoplasty temporarily inadvisable. The positive indications for phrenic nerve interruption are as follows:

1. In conjunction with intrapleural pneumothorax—
 - (a) as an auxiliary measure when marked apical adhesions prevent adequate collapse.
 - (b) as a substitute for pneumothorax when it is impossible to obtain.
 - (c) as an aid during discontinuation of a long-established pneumothorax to prevent overactivity of the expanding lung or serious mediastinal displacement.
2. In the event of a tuberculous effusion or empyema, to help in obliterating the pleural space.
3. In conjunction with total thoracoplasty to collapse the basal part of the lung and save an extra lower stage.
4. In conjunction with pneumoperitoneum to facilitate extreme rise of the diaphragm.

In most instances a temporary crush is performed so that normal respiratory function will be eventually restored. If the disease involves the whole lung or if reopening of collapsed lung is likely to cause reactivation of the disease, a permanent paralysis by avulsion is indicated.

Technique.—The patient is placed on his back with a small pillow under the shoulder and the chin is turned away from the side of operation. The incision for the exposure of the phrenic nerve is made in the neck about 1" above the clavicle at the posterior border of the sternomastoid muscle. The operation is always performed under local anesthesia. The incision is carried down through skin, platysma, and deep cervical fascia. A pad of fat is now encountered which lies on the surface of the scalenus anticus muscle and should be retracted medially by blunt dissection. The scalenus muscle can be felt as a firm flattish cord on

the surface of which the main trunk of the nerve passes downward and medially. When the nerve is gently pinched, the patient usually complains of pain in the shoulder or in the chest. A careful search must be made for accessory branches especially if a temporary or crush operation is contemplated. The nerve, after identification, is injected with local anesthesia and crushed in one place only with a small artery forceps for a temporary result. For permanent paralysis the nerve is divided and avulsed by firm steady traction until it is loosened from its attachments in the thorax; 10 to 12 cm. are removed. Hemostasis is secured and the wound is closed in layers.

Complications.—Injury to adjacent vessels or nerves in the neck, i.e., internal jugular vein, brachial plexus or vagus nerve, has occurred. Occasional shock or intrathoracic bleeding may develop when avulsion is performed. Careful dissection and gentle traction on the nerve is essential to avoid these complications.

Results.—The results of phrenic nerve operations are difficult to assess since operation is generally used as an adjunct to other forms of treatment. The crushing operation paralyzes the diaphragm for 6 to 12 months although rarely a permanent paralysis may result. Avulsion produces a permanent paralysis. These procedures have a definite though limited scope in the surgical treatment of pulmonary tuberculosis.

Thoracoplasty

Introduction.—Thoracoplasty (extrapleural paravertebral) has become the mainstay of the surgical treatment of tuberculosis. The operation has undergone many modifications since its inception in the eighties by de Crenville, Brauer, Friederich, Wilms and Sauerbrück.

The modern operation consists of the extrapleural paravertebral resection of the upper 7th to 8th ribs, performed in stages. The first three ribs are removed completely

around to their costal junctions, and the remaining ones well into the anterior axillary line. The transverse processes of the vertebrae from the 2nd or 3rd down are also resected. The parietal pleura is stripped downward at the apex (apicolysis) to depress the apical portion of the lung to the level of the hilus. This procedure allows the lung to retract medially and concentrically, and the collapse is maintained by the permanent reduction in the size of the hemithorax. The principles of healing—granulation and fibrosis—bring about closure and healing of cavities in the permanently collapsed lung in a great majority of cases, although this process may take many months or even years. The test of success is the closure of cavities and the disappearance of bacilli from the sputum.

Indications.—The pre- and postoperative use of antibiotics (dihydrostreptomycin and para amino salicylic acid) has greatly increased the scope of major operative procedures in surgical tuberculosis. Many cases that were previously considered inoperable due to the extent of the disease (bilateral) or to poor general condition have been successfully operated upon following the use of these drugs. The usual indications for thoracoplasty were in the so-called "good chronic" patient with unilateral cavitary tuberculosis, in whom less radical forms of treatment (pneumothorax, sanatorium care) had failed to arrest the disease. However, the term "good chronic" does not apply to patients with long-standing disease and thick-walled indurated cavities, since this type does not respond well to thoracoplasty, due to the indurative nature of the process.

The condition of the contralateral lung is an important factor in the decision to perform thoracoplasty. Small fibroid or calcified foci in the apex can be disregarded. Apical thoracoplasty, 1 to 4 ribs, may be contemplated even if the "good" lung shows arrested tuberculosis of moderate degree. However, the presence of recent active dis-

case in the contralateral lung is regarded as an absolute contraindication to thoracoplasty. Occasionally the institution of a selective pneumothorax for several months on the less involved side may be performed in preparation for thoracoplasty on the other.

Contraindications.—The contraindications to thoracoplasty apart from recent exudative disease in the opposite lung are those arising from the general state of the patient. Patients with inadequate cardiac, respiratory, or renal reserve are poor surgical risks for thoracoplasty.

done in stages; 3 to 4 ribs are resected at each stage according to the general condition and resistance of the individual patient.

Preoperative Preparation and Anesthesia.—The properly selected and investigated case is given preoperative medication as required by the choice of anesthetic agent. Local anesthesia is used by a few operators and requires heavy sedation either with barbiturates or morphia. High spinal (Nupercaine) has distinct advantages for chest surgery but requires an anesthetist adequately trained in its use. It allows the patients to

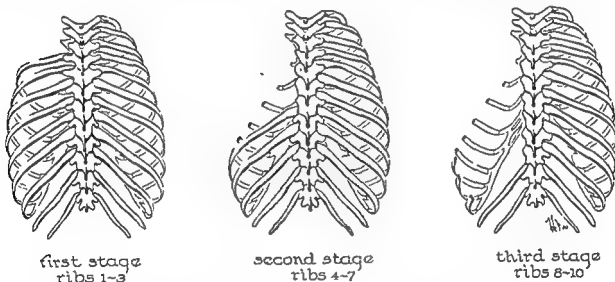


Fig 144.—Number and extent of ribs resected in a three-stage posterolateral thoracoplasty.

The estimation of vital capacity and in doubtful cases bronchspirometry should be performed before any major collapse operation is undertaken. Age is not a contraindication, per se, if the general condition is adequate, and successful thoracoplasty has been performed in the 60 to 65 year age group. The operation is well tolerated by children when indicated. Patients with the signs of an acute reaction—elevated temperature, pulse and sedimentation rate—should not be subjected to operation until several weeks or months after return to normal.

Technique.—The modern posterolateral thoracoplasty with or without apicolysis is

retain their cough reflex and greatly diminishes the shock relative to rib resection. Moderate preoperative sedation is essential. The most usual type of anesthesia used is some form of endotracheal anesthesia, providing the ability to aspirate the bronchial tree during and at close of the operation. Nitrous oxide and oxygen and Cyclopropane are the usual agents

Position of Patient and Incision.—Most operators prefer the posterolateral position of the patient with the arm of the affected side abducted over the head to facilitate mobilization of the scapula. Some use the prone position on a special table (Overholt). The conventional incision starts 1½ to 2"

from the spinous processes at the level of the 2nd dorsal spine and sweeps downward and laterally following the angle of the scapula out to the posterior axillary line.

First Stage Operation.—Following the above incision the third rib is identified, the periosteum stripped, and the rib removed well around in the axillary line (it can frequently be excised at its chondral junction). This facilitates the removal of the 2nd and 1st ribs in this order. These two ribs are disarticulated at the sternum if possible.

As a rule only three ribs are removed at this session, occasionally the 4th may be added if the condition of the patient is good, and there is no tendency to paradoxical respiration. If apicolysis is desirable, it is performed by utilizing the plane of the endothoracic fascia to free the apex of the lung, depressing it downward toward the hilus.

The transverse processes of the 2nd, 3rd, and 4th thoracic vertebrae are resected to aid in the compression of cavities which may be displaced posteriorly. The wound is closed in layers without drainage. Postoperatively the patient is watched for shock. Oxygen is given for respiratory distress. Morphine is administered cautiously in order to avoid depression of the cough reflex. Tracheal section or bronchoscopy should be employed if necessary.

A period of 2 to 3 weeks is the usual length between stages. Subsequent stages are performed as required in the individual case. The previous incision is reopened (the scar is excised by some surgeons) and the next 3 or 4 ribs are resected as far as the anterior axillary line, the transverse processes included. There may be more paradoxical respiration following the lower stages. Occasionally an anterolateral stage may be required to remove the anterior ends of the ribs from the 3rd down in order to complete the collapse of the upper part of the thorax. A 7 or 8 rib thoracoplasty allows the scapula to fall against the chest wall adding to the compressing force. The tip of the scapula can be resected in a 5 or 6 rib thoracoplasty

to achieve the same result. A total thoracoplasty requires a third posterior stage including the 9th or 10th rib.

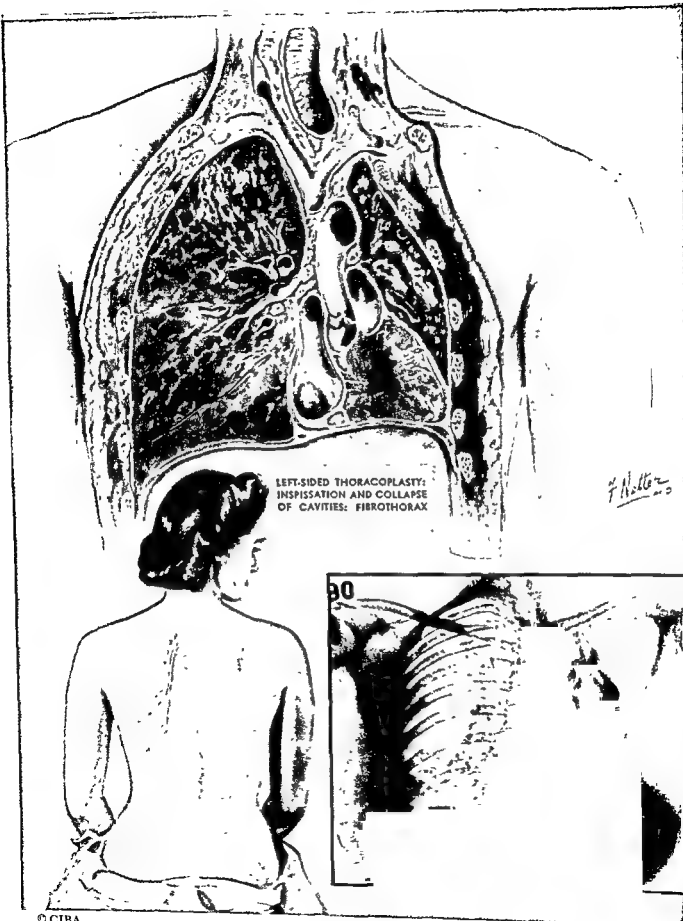
Complications may be classified as follows:

1. *Operative.*—Operative shock and hemorrhage are controlled by proper anesthesia and adequate blood replacement during operation, as well as by limiting the amount of rib resection at each stage to the resistance of the individual patient.

2. *Functional Disturbance.*—Cardiorespiratory embarrassment may result from too rapid compression of the lung in patients with emphysema or impaired function of the contralateral lung due to disease or fibrosis. Paradoxical respiration, i.e., indrawing of the operated side during inspiration may also cause cardiorespiratory distress.

3. *Postoperative Spread of Infection.*—Spread of tuberculous infection to the good lung or base of the operated side occasionally occurs despite all precautions to prevent it. Wound infection, pyogenic or tuberculous, is now a rarity with the adequate preoperative and postoperative use of antibiotics.

Results.—The success of thoracoplasty operation is judged by the closure of cavities and the disappearance of bacilli from the sputum, as well as by the ability of the patient to return to partial or complete economic independence. A postoperative convalescent period of 6 months to one year is necessary to consolidate the healing process facilitated by the operation. It has been estimated that about 75% of thoracoplasty patients have arrested disease and are able to carry on normal activities; 15% continue to have active disease, and 7 to 8% are early or late postoperative deaths. Reasons for failure of arrest of disease after thoracoplasty include tuberculous bronchitis and bronchiectasis, the so-called tension or valvular cavities which resist compression and may continue to expand after operation, and activation of disease in uncollapsed areas of the lung.



LEFT-SIDED THORACOPLASTY:
INSPISSATION AND COLLAPSE
OF CAVITIES: FIBROTHORAX

F. Noller
1935

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CHAPTER XV

HEART AND PERICARDIUM

CHARLES B. RIPSTEIN, M D

Introduction.—Cardiac surgery has made tremendous strides in the past ten years. This advance has been due to a better understanding of the physiology of the heart and to improved methods of anesthesia and postoperative care. It is now realized that the chief causes of mortality in cardiac operations are insufficient oxygenation of the blood, mechanical displacement of the heart, and irritation of the myocardium with the development of ventricular fibrillation. The additional factors of shock, hemorrhage and embolization are also important. We now have the means of preventing and treating most of these conditions. The use of the oximeter and positive pressure intratracheal anesthesia control oxygenation. Modern surgical approaches obviate the need for displacing the heart. The value of procaine and its derivatives in preventing fibrillation is now well recognized, and the electric defibrillator has proved its value in the established case.

Many diseases of the heart are now amenable to surgical therapy and new conditions are rapidly being added to the list. Cardiac surgery is a well-established field and can no longer be considered a curiosity without practical importance.

TRAUMA TO THE HEART

The earliest form of heart surgery involved the treatment of wounds, and although these were almost universally fatal, it was on this basis that cardiac surgery developed. In considering trauma to the heart, three types of injury are important:

1. Contusions.
2. Penetrating wounds.
3. Foreign bodies within the heart or its chambers.

Contusions of the heart are common in modern times, exemplified by the so-called "steering-wheel injury." The cause is usually an automobile collision resulting in a severe blow over the precordium. There may or may not be an associated injury of the thoracic cage.

The initial symptoms are weakness, pain, dyspnea and cyanosis. Rarely, rupture of the heart may occur with signs of massive hemorrhage and shock. The electrocardiogram shows transient S-T changes which revert to normal within a few days.

Contusions of the heart are often overlooked and spontaneous recovery usually occurs. However, in some cases extensive myocardial damage may be produced. For this reason, the possibility must be borne in mind and electrocardiograms should be routine in all cases of severe contusion of the chest or upper abdomen.

Penetrating wounds of the heart are caused by stabbing or bullets. The symptoms are due to massive hemorrhage and acute cardiac compression. The wound of entry usually overlies the heart but may be in the abdomen or neck.

Most wounds of the heart demand immediate surgical intervention. If the instrument causing the injury is protruding through the chest wall, it should not be removed because it may act as a tampon and prevent fatal hemorrhage. The essentials in the treatment of cardiac wounds are arrest of bleeding, replacement of blood loss, and release of cardiac compression.

The heart is approached through an anterior incision in the fourth interspace. When the pericardium has been opened, the wound may be temporarily plugged with the surgeon's finger while through-and-

through mattress sutures of silk are passed about the laceration. The heart muscle tears easily and should not be grasped with instruments. The finger is withdrawn as the sutures are tied, and in this way, the bleeding is arrested. A gelatin sponge pad may be used to reinforce the suture line. The mortality in penetrating cardiac wounds is high, but many lives can be saved by prompt surgical intervention. Recently, Ravitch and Blalock have advocated conservative treatment for cardiac wounds. They point out that if cardiac compression is relieved by pericardial aspiration the bleeding usually ceases spontaneously. This has proved to be a rational concept and has been borne out by personal experience.

Foreign bodies in the heart occur as a result of bullets or shrapnel wounds and are rarely seen in civilian life. They give rise to complications in 50% of cases, and in these patients, their removal becomes mandatory. The complications are:

1. Emboli.
2. Ulcerative endocarditis.
3. Cardiac abscess.
4. Rupture of the heart wall with hemorrhage.
5. Cardiac aneurysm.
6. Psychic disturbances due to the patient's knowledge of the presence of a foreign body in his heart.

The removal of foreign bodies can be carried out with a low mortality. It is recognized, however, that sterile foreign bodies rarely cause symptoms, and operation should not be undertaken without a definite indication.

CARDIAC COMPRESSION

Compression of the heart (tamponade) interferes with cardiac action by preventing adequate ventricular output and venous return. In the acute form, death occurs if the tamponade is not relieved. The heart will tolerate a considerable degree of chronic compression, but inevitably cardiac failure occurs.

Acute compression is always due to fluid, either exudate or blood. The former is a manifestation of acute pericarditis, the latter of cardiac trauma. The clinical syndrome is similar in both.

The characteristic signs of acute cardiac compression are found, in the "Beck triad":

1. A small quiet heart.
2. Rising venous pressure.
3. Falling arterial pressure.

The presence of these signs establishes the diagnosis of cardiac tamponade and unless the pressure is relieved, death will result. Ravitch and Blalock have pointed out that pericardial aspiration is the treatment of choice. Pericardiostomy is rarely necessary.

Chronic compression may be produced by fluid, scar tissue or rarely by tumor. The heart can tolerate gradual compression for a fairly prolonged period, but ultimately failure results. Chronic compression impairs heart action in two ways: by preventing adequate venous return, and by interfering with cardiac output. The commonest cause is chronic constrictive pericarditis which is usually due to tuberculosis.

The clinical syndrome of chronic compression is exemplified by the second Beck triad of:

1. A small quiet heart.
2. Elevated venous pressure.
3. Enlarged liver with ascites.

X-ray reveals a small heart shadow with little or no pulsation on the kymograph film. Calcification may be present in the pericardium. The electrocardiograph shows low amplitude in all leads, but no specific abnormalities. Other investigation confirms the fact that cardiac function is impaired; the circulation time is prolonged, exercise tolerance is poor, and dyspnea and cyanosis are usually present. The treatment consists of removal of the compressing scar by pericardiectomy. The best approach for this procedure is through a midline sternum-splitting incision. In this way both ventricles may be freed in one operation. Following adequate decompression, all symp-

toms disappear and the cardiac function returns to normal. Recently, Holman has advocated pericardiectomy in the acute phase of tuberculous pericarditis. He feels that the crippling late manifestations of the disease may thus be forestalled and there is good evidence that this is a worth-while procedure.

from a few pioneers no one became very much concerned about these children.

The development of cardiac surgery in the past ten years has changed the picture completely. The field of congenital anomalies is obviously a fertile one, for in most cases the myocardium is healthy, and if the anatomical or functional abnormality can be cor-

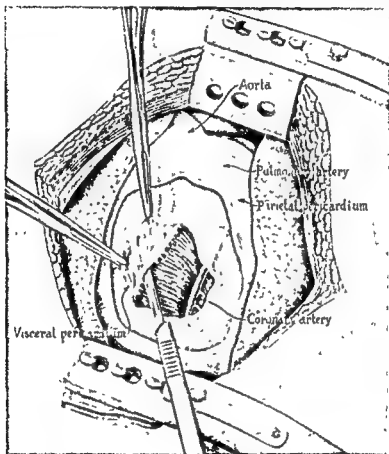


Fig 145—Chronic constrictive pericarditis Separation of thickened pericardium from the Heart

CONGENITAL ANOMALIES

Until recently, the differential diagnosis of the various types of congenital heart disease was a matter of academic interest, but of little practical importance. No definitive treatment was available and although a few clear-cut entities could be clinically recognized, the diagnosis was only confirmed at autopsy. As a result, little interest was aroused in cardiac anomalies, and apart

rected, a beneficial result may be expected. The first major advance was made by Robert Gross in 1939, when he reported the successful ligation of a patent ductus arteriosus. With this stimulus, interest in congenital cardiac anomalies became more widespread. The development of new diagnostic aids has made possible an exact determination of the anatomical and physiological disturbances in each case. New methods of anes-

thesia have made it feasible to carry out delicate and prolonged operations. New techniques in vascular surgery have been perfected. These advances have resulted in procedures which were undreamed of a few years ago.

Classification of Congenital Heart Disease

Many types of congenital lesions of the heart have been described. When we consider the complex embryology of this organ, it is surprising that anomalies do not occur even more frequently. A practical classification which is of value in diagnosis and therapy is the following:

I. ACYANOTIC GROUP

Anomalies in which there is no interference with oxygenation of the blood. Pulmonary blood flow is usually normal or may be increased. Symptoms are produced by the extra mechanical strain imposed on the heart or by pressure on neighboring organs.

- A. *Patent Ductus Arteriosus.*
- B. *Anomalies of the Aortic Arch.*
 - i. Double arch.
 - ii. Anomalous origin of subclavian or innominate arteries
- C. *Coarctation of the Aorta.*
- D. *Aortic Stenosis.*
- E. *Septal Defects.*
 - i. Auricular.
 - ii. Ventricular.
- F. *Anomalous Origin of the Coronary Arteries.*

II. CYANOTIC GROUP

Pulmonary blood flow and oxygenation of the blood are deficient. The symptoms are produced by chronic anoxia, as well as by the extra strain imposed on the heart.

- A. *Tricuspid Atresia with Failure of Development of the Right Ventricle.*
- B. *Tetralogy of Fallot.*
- C. *Pulmonary Stenosis.*
- D. *Transposition of the Great Vessels.*
- E. *Truncus Arteriosus.*

Many others have been described, but in this discussion we will confine ourselves to those congenital cardiac defects which can be successfully treated by operation.

Patent Ductus Arteriosus.—The ductus arteriosus is normally open during fetal life. It conveys blood from the pulmonary artery to the aorta, short-circuiting the pulmonary circulation, which is unnecessary while the placenta functions. At birth, when the infant begins to breathe, the channel is closed by muscular contraction of its walls and during the next few months it gradually obliterates to form the fibrous ligamentum arteriosum. In about 1% of individuals, the ductus remains patent and this results in a permanent fistula between the aorta and the pulmonary artery.

The effects of a patent ductus depend on the size of the opening and the degree of shunt through it. If this is small, it may be well tolerated and the patient live a normal life free from symptoms. In most cases, one or more of the following result:

1. Because of the higher aortic pressure, blood is deviated from the peripheral circulation into the pulmonary circuit. The oxygen content of the blood is higher than normal, but an inadequate volume flow results and the child may be small and poorly developed.

2. Cardiac hypertrophy occurs from the strain of compensating for the abnormal fistula. Congestive heart failure is the end result, usually occurring in the third decade.

3. Subacute bacterial endocarditis may arise with the formation of vegetations at the site of the ductus.

4. Due to the mechanical weakness, aneurysm may occur at the site of the ductus. This frequently ruptures with fatal hemorrhage. Because of these grave complications, the patent ductus constitutes a serious danger to life, and Keyes and Shapiro have estimated that the life expectancy of individuals with a patent ductus arteriosus is about half that of the population in general.

The diagnosis is usually made on the basis of clinical findings. The child is undersized and pale, but there is no cyanosis. The heart may be enlarged and characteristically a loud rumbling biphasic murmur is heard in the second left interspace. This murmur

Laboratory tests are of some help. The oxygen content of the arterial blood is high. The electrocardiogram may show left axis deviation, and as Gross points out, right axis deviation is never seen with an uncomplicated ductus.

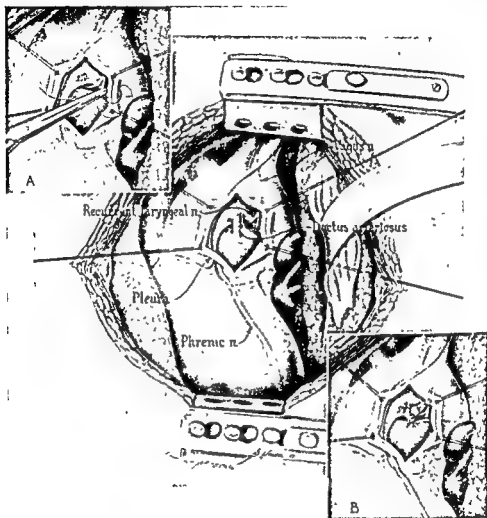


Fig 146.—Patent ductus arteriosus. Inserts A and B demonstrate surgical division and ligation of the ductus.

has a peculiar "machinery" quality and it may be transmitted into the neck, axilla, and scapular region. In most cases a palpable thrill is present over the pulmonic area. The blood pressure shows a normal or slightly elevated systolic and a low diastolic similar to the findings in any other large arterio-venous fistula.

X-ray examination gives very important information. The heart shadow is often enlarged and the pulmonary artery is prominent. The hilar markings are accentuated and pulsation may be visible on fluoroscopy. Cardiac catheterization can be used to confirm the diagnosis and rule out associated abnormalities, but it is unnecessary in most

cases, except where the exact volume of the shunt must be determined.

The treatment of choice is surgical ligation or division of the patent ductus. The heart is approached through the left pleural cavity and the ductus arteriosus is visualized and mobilized. The channel can be obliterated by multiple ligatures or divided between clamps and the ends oversewn. The latter procedure is more hazardous, but insures complete and permanent occlusion of the ductus. Postoperatively, the murmur disappears, the diastolic pressure rises and the heart decreases in size. The patient then has a normal heart and a full life expectancy.

In children, the mortality is low (2 to 3%) and the results are excellent. In adults, the operation is somewhat more hazardous and should not be recommended unless the shunt is large.

If any degree of cyanosis is present, the ductus should not be ligated because it may serve as a compensatory shunt for associated cardiac abnormalities.

Abnormal Aortic Arch.—Several anomalies fall into this group: double aortic arch, and abnormal origins of the innominate and subclavian vessels. The result in all these cases is a vascular ring formed by the aorta and its abnormal branches. This surrounds the esophagus and trachea and produces symptoms due to pressure on these structures.

The classical syndrome of "dysphagia lusoria" is caused by esophageal obstruction. Dyspnea, cyanosis, stridor and recurrent pulmonary infections may occur with tracheal obstruction. The cause of the obstruction can be determined by fluoroscopic examination. A pulsating defect in the trachea and in the barium-filled esophagus is the characteristic finding.

The treatment is exploration and division of the constricting vascular ring. As a rule, the anterior limb is divided and the posterior limb is left intact.

Coarctation of the Aorta.—This condition is a congenital narrowing of the thoracic aorta. It usually occurs at the attachment of the ligamentum arteriosum, and some believe that coarctation represents a continuation of the obliterative process which normally occurs in the ductus.

Two pathological varieties have been described:

1. Infantile type, in which the narrowing occurs over a long segment. This is usually associated with other abnormalities and is not as a rule compatible with prolonged life.

2. Adult type, in which the constriction is localized to an area one centimeter in length or less.

The signs and symptoms of coarctation of the aorta depend upon the obstruction to the blood flow and the compensatory phenomena necessary to overcome it. This results in:

1. Hypertension in the upper part of the body.

2. Low blood pressure and absent arterial pulsation in the lower extremities.

3. Cardiac enlargement.

4. Development of collateral circulation, enlargement of scapular vessels, intercostals (notching of ribs) and internal mammaries.

Complications occur due to the hypertension and mechanical strain at the narrowed area. The most dangerous of these are:

1. Cerebral hemorrhage.

2. Cardiac failure.

3. Rupture of the aorta.

4. Subacute bacterial endocarditis.

Individuals with coarctation rarely live past the third decade and usually succumb to one of the above complications.

Crafoord first reported the successful resection of a coarctation with end-to-end suture of the aorta. Gross, Blalock, and others have since performed this operation on many patients, and, especially in the younger age group, it is an entirely feasible procedure. Correction of the coarctation restores the circulation to normal.

The diagnosis is usually made on the basis of clinical findings. The child is undersized and pale, but there is no cyanosis. The heart may be enlarged and characteristically a loud rumbling biphasic murmur is heard in the second left interspace. This murmur

Laboratory tests are of some help. The oxygen content of the arterial blood is high. The electrocardiogram may show left axis deviation, and as Gross points out, right axis deviation is never seen with an uncomplicated ductus.

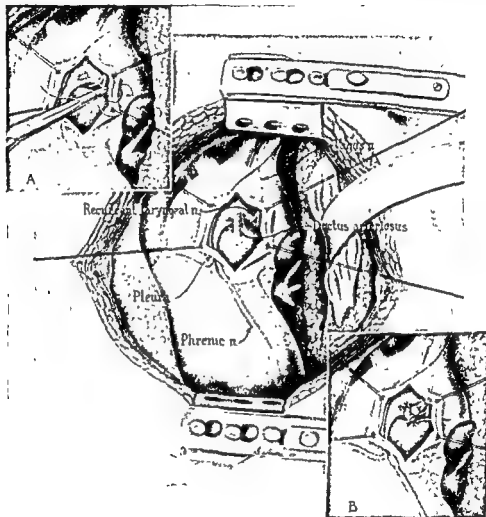


Fig 146.—Patent ductus arteriosus. Inserts A and B demonstrate surgical division and ligation of the ductus.

has a peculiar “machinery” quality and it may be transmitted into the neck, axilla, and scapular region. In most cases a palpable thrill is present over the pulmonic area. The blood pressure shows a normal or slightly elevated systolic and a low diastolic similar to the findings in any other large arterio-venous fistula.

X-ray examination gives very important information. The heart shadow is often enlarged and the pulmonary artery is prominent. The hilar markings are accentuated and pulsation may be visible on fluoroscopy. Cardiac catheterization can be used to confirm the diagnosis and rule out associated abnormalities, but it is unnecessary in most

defects, but to date, no operation has been evolved which is feasible in the human being.

Tetralogy of Fallot.—Fallot originally described a group of anatomical abnormalities which he found in many cases of congenital heart disease with cyanosis. Abbott con-

2. Right ventricular hypertrophy.
3. Dextroposition of the aorta.
4. Pulmonary artery stenosis.

Other associated lesions are sometimes present.

Besides the extra strain imposed on the heart, the effect of these anatomical ab-

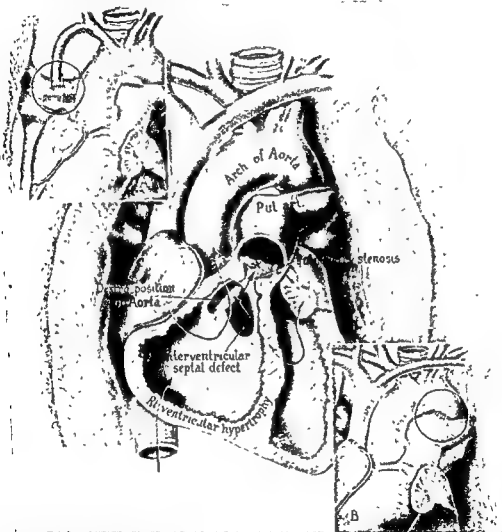


Fig. 148.—Tetralogy of Fallot.

Insert A shows the Blalock procedure of anastomosis of the subclavian artery to the right pulmonary artery.

Insert B shows Pott's operation of direct aortopulmonic anastomosis.

firmed the fact that the tetralogy of Fallot represents the most common cause of cyanotic heart disease. The lesions comprising this tetralogy are:

1. Interventricular septal defect.

normalities is to cause a mixture of left and right heart blood and to prevent sufficient blood from reaching the pulmonary circulation. The result is deficient oxygenation of the blood and consequently cyanosis, club-

Blalock has suggested an alternative, anastomosis of the left subclavian artery to the aorta distal to the narrowed segment. This procedure has been employed successfully in cases where the coarctation is too long to permit end-to-end suture. Recently, Gross has reported the successful use of preserved

not possible and the aim of operation is to improve the function of the heart despite existing anomalies.

Septal Defects.—Congenital defects may occur in the interauricular or interventricular septa. In some cases, these do not constitute a serious disability, but if the defect

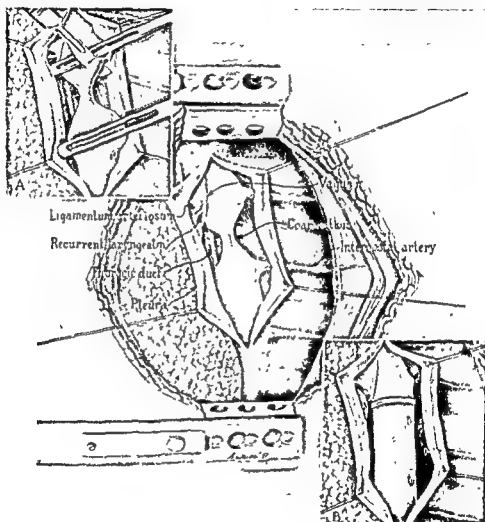


Fig 147—Coarctation of the aorta The technique of resection and anastomosis is shown in the inserts.

arterial grafts to replace segments of aorta excised for coarctation. The ultimate fate of such grafts has not been determined, but the immediate results are excellent

In all of the above conditions, surgical operation restores the circulation to normal. There are many anomalies in which this

is large, there may be sufficient shunt of blood to embarrass the circulation.

The diagnosis can be made by x-ray, but cardiac catheterization is necessary to rule out associated abnormalities

A great deal of experimental work has been done on the surgical closure of septal

pectancy, but as Blalock points out, the fear of some indefinite future complication is insufficient reason for permitting these children to die from anoxia.

Pulmonary Artery Stenosis.—Pulmonic stenosis may exist as an isolated lesion but is usually associated with a patent foramen ovale. The narrowing of the pulmonary orifice results in deficient blood flow through the lungs. Cyanosis is not always present at rest, but appears on exertion and exercise tolerance is poor. Characteristically, there is a harsh systolic murmur over the pulmonic area and on x-ray the pulmonary artery is dilated. Cardiac catheterization reveals hypertension in the right ventricle with an abrupt fall beyond the stenotic valve.

The treatment of this lesion is by pulmonary valvulotomy which relieves the stenosis without adding an artificial shunt. Brock, Bailey, and others have devised instruments to cut and dilate the pulmonary valve and the results of operation have been very satisfactory. The Blalock procedure is contraindicated in these cases because it does not attack the essential anomaly and although a shunt may relieve the cyanosis, it imposes an additional strain on the heart.

Transposition of the Great Vessels.—In this complex anomaly, the aorta arises from the right ventricle and the pulmonary artery from the left. If this arrangement is to be compatible with life, some mixture of blood must occur between the two sides of the heart. This may be accomplished through a patent ductus, a septal defect, or both. The surgical treatment involves the artificial creation of such shunts. To date, the results have been encouraging, but the mortality rate is high.

ACQUIRED VALVULAR LESIONS

Mitral Stenosis

The idea of surgical treatment for mitral stenosis is not new. Brunton discussed the problem in detail 50 years ago, and in 1925 Souttar reported a successful case of digital

dilatation of the mitral ring. It is difficult to understand why this method of treatment was not widely applied, but the unfortunate results of valvulotomy reported by Cutler and Beck in 1929 undoubtedly discouraged many workers in this field. They felt that it was necessary to excise a portion of the mitral valve and thus create a mitral regurgitation in place of the stenosis. In many cases, stenosis is better tolerated than insufficiency so it is obvious that bad results would follow such a procedure. The concepts of valvuloplasty introduced by Harkin and commissurotomy by Bailey and his associates have made it possible to correct the deformity of mitral stenosis without removing valve tissue and creating a regurgitation. These workers pointed out that the chief area of fusion in mitral stenosis is along the commissures between the cusps, and that if this line of fusion is cut, a functioning mitral valve will be produced with little or no regurgitation. Special knives have been devised which enable the surgeon to engage and cut the commissures through an incision in the left auricular appendage. The results in a large series of cases have been very encouraging, about 65% of patients benefiting from the operation. The chief dangers are the creation of a major degree of regurgitation and the formation of emboli from thrombus in the auricle or its appendage.

Aortic Stenosis

Smithy has recently attempted valvulotomy of the aortic valve with an instrument introduced through the left ventricle. This procedure removes a piece of the stenotic valve and substitutes an insufficiency for a stenosis. To date the results are not encouraging.

CORONARY ARTERY DISEASE

Coronary artery disease is becoming more prominent as a cause of death and disability. It is primarily a degenerative disease and its ultimate cure probably lies in the field of metabolism and nutrition. However, at-

bing of the fingers, polycythemia and decreased exercise tolerance. The severity of symptoms depends on the degree of pulmonary flow present. These children are underdeveloped and cannot tolerate physical activity. Their life expectancy is short and death occurs because of cardiac failure or cerebral thrombosis.

Several types of shunt have been used. Anastomosis of the right or left subclavian artery to the pulmonary is probably the most satisfactory method. The innominate or carotid arteries can be used, but there is a high incidence of cerebral complications.

Potts and his co-workers have anastomosed the pulmonary artery directly to the aorta

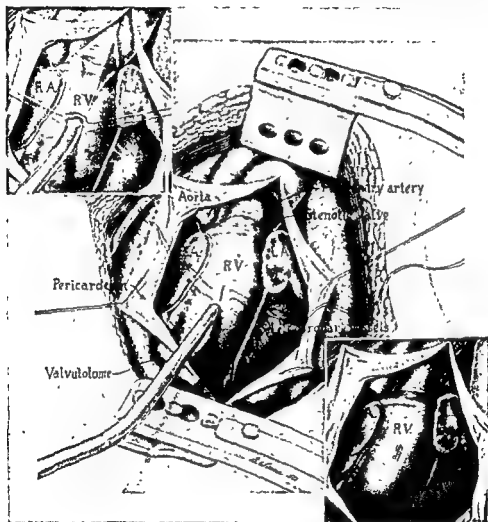


Fig 149.—Pulmonic stenosis. Technique of valvulotomy

Taussig pointed out that because of the anatomical anomalies present, the essential feature of this lesion is the failure of adequate oxygenation of the blood. She and Blalock devised the shunt operation which permits the transfer of adequate amounts of arterial blood into the pulmonary circuit.

with excellent results. The principle of all these approaches is similar and the results in all have been initially good. It is true that the essential abnormality of the heart remains unchanged, and the shunt imposes an additional strain. It is still too early to speak of the end results in terms of life ex-

the coronary tree. Ordinarily such communications are small, but in cases of coronary occlusion, they may contribute a large share of the blood reaching the heart capillaries.

The various methods employed in attempts to revascularize the myocardium may be summarized as follows:

1. *Development of collateral circulation by vascular grafts.*

3. *Development of existing anastomotic channels within the heart.*

(a) Coronary sinus ligation (Gross, Blum, and Silverman).

(b) Great cardiac vein ligation (Fau-teux).

4. *Arterialization of the coronary veins (Beck).*

It is difficult to assess the results of any procedure designed to improve the coronary

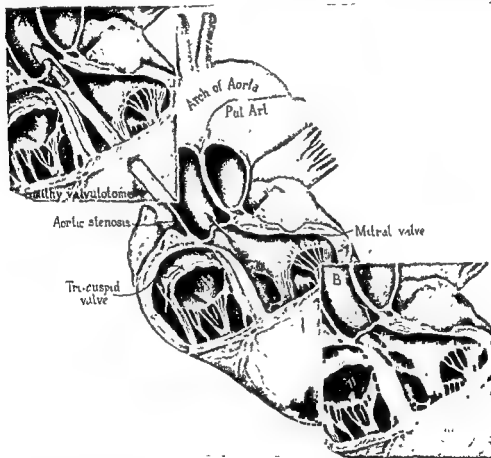


Fig 151.—Aortic stenosis Smithy valvulotome

- (a) Pectoral muscle (Beck).
- (b) Omentum (O'Shaughnessy).
- (c) Internal mammary artery (Vineberg).

2. *Development of collaterals from cardio-pericardial adhesions.*

- (a) Asbestos (Beck)
- (b) Aleuronat (Heinbecker).
- (c) Talc (Thompson).

circulation. All of the operations have been carried out on patients with severe angina of effort and the criteria used by most authors to evaluate results are:

- 1. Freedom from anginal pain
- 2. Increased ability to work.
- 3. Period of survival.

It is extremely difficult to evaluate a procedure on the basis of subjective phenomena,

tempts are being made to improve the blood supply to the diseased heart by surgical means.

The concept that the coronary vessels are end arteries must be discarded. Prinzmetal has shown that communications exist between the various branches of the coronary arterial tree and also between arteries and

the excised mammalian heart could maintain contractions on blood perfused through the coronary sinus even though no arterial flow was present. Many human cases have been reported in which the nutrition of the heart was adequate even though both coronary arteries were occluded. It appears then, that there are sufficient collateral channels

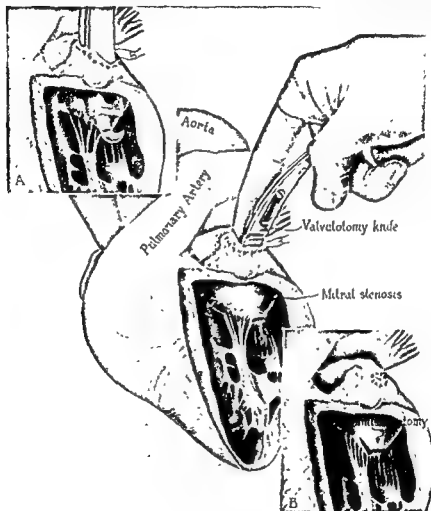


Fig 150—Mitral stenosis The technique of mitral commissurotomy.

veins. In pathological conditions, the capillary bed in the myocardium can receive blood from the arteries, the veins, or from the heart chambers via the Thebesian vessels. Since there are no valves in these vessels the direction of flow may be in any direction depending on the pressure gradients established. It had long been known that

to maintain the nutrition of the heart even when the arterial flow is cut off. These apparently do not function adequately in many clinical cases of coronary artery occlusion.

In addition to the channels within the heart itself, extracoronary collaterals exist in the mediastinal, bronchial, and pericardial vessels, and these can form connections with

and until we have a feasible means of recording myocardial blood flow before and after operation, it will be impossible to determine benefit from any of the suggested procedures.

To date, all operations for coronary disease have carried a high mortality, and in none have the results been convincingly beneficial. The whole subject requires a great deal more experimental work before a wide application to human material can be justified.

TUMORS OF THE HEART

Primary tumors of the heart and pericardium are rare but can occasionally be diagnosed by x-ray and treated by surgical excision. They are usually fibromas, lipomas or rhabdomyomas.

Metastatic tumors occur in the heart, but are of no surgical significance.

CARDIAC RESUSCITATION

Cardiac arrest represents one of the most dramatic situations in surgery and nowhere is prompt treatment more essential. It is estimated that cardiac arrest occurs once in 1,500 to 2,000 operations, so it is obvious that these cases will be encountered several times a year in most clinics.

Cardiac arrest in the surgical patient may be due to cardiac standstill or to ventricular fibrillation. The differential diagnosis is of vital importance because the treatment differs in the two conditions. The diagnosis is often difficult and requires confirmation by an electrocardiographic tracing. If the heart can be palpated or visualized, the condition of ventricular fibrillation can be recognized by the uncoordinated twitching of the ventricular muscle.

The treatment of cardiac standstill involves stimulating the myocardium with adrenalin and cardiac massage. The latter procedure is of value in two ways. First, it enables the operator to force blood from the heart into the great vessels, and this,

with the effect of artificial respiration, gives enough circulation to the brain and other vital organs to keep the patient alive until coordinated heart action is resumed. Second, massage provides a good irritative stimulus to the myocardium which will often initiate the beat.

The proper technique of cardiac massage is a controversial matter. Manipulation through the diaphragm is of little value and cannot be used to maintain circulation for any time. If the abdomen is open when the heart stops, the left side of the diaphragm may be incised and the hand introduced into the thoracic cavity. The heart is palmed and rhythmic compression and relaxation begun. The heart is compressed from the apex to the base in order to expel blood through the aortic orifice. The trans-thoracic approach is preferable because the heart can be visualized and observed more accurately. An intercostal incision in the 5th left interspace allows rapid exposure. The technique of cardiac massage should be learned by all surgeons. If it is effectively done the radial, femoral and carotid pulses become palpable and circulation can be maintained for an indefinite period.

Two other methods are available which aid in maintaining an effective blood flow during cardiac arrest. Heparin is of value in preventing thrombosis and keeping the blood fluid, and the tilt table has proved to be an effective method of aiding circulation.

The use of adrenalin in cardiac arrest has been much abused in the past. It is a very valuable drug in cases where the heart has stopped, but it is dangerous in too large a dosage because it may precipitate ventricular fibrillation. The dose should not exceed 0.5 ml of the 1:1,000 solution and should be given into the left ventricle. In the presence of fibrillation its use is contraindicated because it increases the irritability of the myocardium.

Ventricular fibrillation can no longer be considered a hopeless condition. Cases have

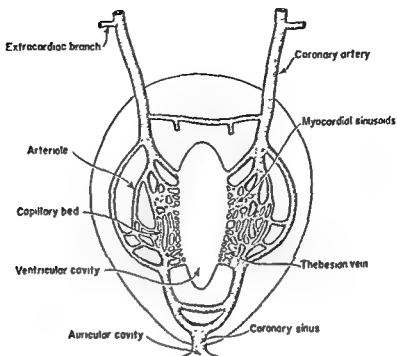


Fig. 152—Collateral circulation of the heart

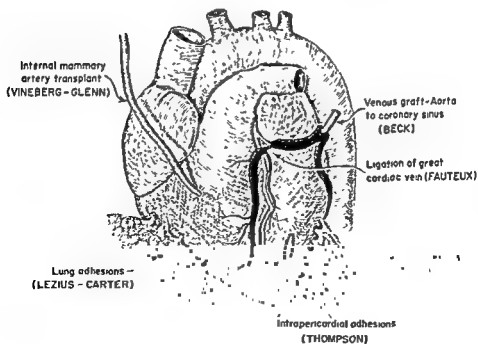


Fig. 153.—Procedures for revascularization of the myocardium.

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Ventricular fibrillation can no longer be considered a hopeless condition. Cases have

been reported by Beck and by Lampson in which the human heart has been successfully defibrillated. These experiences confirm the experimental work of Beck, Fauteux and others. The technique of treatment is now well established and many more successful cases will undoubtedly be reported in the future. The first requisite is cardiac massage and artificial respiration to maintain an adequate circulation of oxygenated blood. The next step is the restoration of a coordinated heartbeat.

Procaine hydrochloride in dosage of 5 c.c. of a 1% solution should be given intravenously or directly into the heart. In some cases, this alone will cause a coordinated action of the ventricles. In most instances, a second dose is necessary followed by electrical stimulation. This is done by using ordinary 110 volt alternating current with 15 amperes. An electrode is placed on either side of the heart and the current turned on for 1 or 2 seconds. The effect is a simultaneous contraction of all the heart muscle fibers. This results in cardiac standstill which is followed by a regular coordinated rhythm. Several shocks may be necessary before this is attained.

The human heart has been successfully defibrillated several times with complete recovery of the patient. The time element is important, because if a state of cardiac standstill is allowed to persist for longer than three to four minutes irreversible brain damage occurs. Other vital organs such as the liver, kidneys, and heart itself also suffer from prolonged anoxia. It is obvious that if these resuscitative measures are to be of value they must be applied without delay.

ARTIFICIAL HEART-LUNG MACHINES

In the past few years, a great deal of experimental work has been done in developing a mechanical apparatus which will

temporarily take over the functions of the human heart and lungs. There are two potential fields of use for such a machine: first to relieve a failing heart and permit its recovery from an acute insult, and second to render the heart bloodless for a period and thus permit the surgeon to operate within its chambers under direct vision. Gibbon, Dennis, and others have devised machines which successfully perform the functions of the heart and lungs in the experimental animal. To date, the trials in human cases have been unsuccessful, but there is no doubt that the technical difficulties will be solved in the near future and it will be possible to attack many cardiac lesions by a direct approach.

The various types of apparatus used all work on similar principles. Blood is deviated from the venae cavae, pumped through an oxygenator and returned into the aorta depending on a competent aortic valve to prevent regurgitation into the heart. The animal must be heparinized to prevent clotting in the extracorporeal circuit.

REFERENCES

- Bailey, C. P., Lacy, M. M., and Harris, J. S. C.: The Surgical Treatment of Acquired Heart Disease, *Surg. Clin North America* 31: 1821-1863, Dec., 1951.
- Beck, C. S.: Principles Underlying the Operative Approach to the Treatment of Myocardial Ischemia, *Ann. Surg.* 118: 788-806, 1943.
- Bellet, S.: Diagnostic Features of Cardiovascular Lesions Amenable to Surgery, *Surg. Clin North America* 31: 1801-1809, Dec., 1951.
- Blalock, A.: A Consideration of Some of the Problems in Cardiovascular Surgery, *J. Thoracic Surg.* 21: 543-571, 1951.
- Gross, R. E.: Surgical Treatment for Abnormalities of the Heart and Great Vessels, *American Lecture Series*, Springfield, Ill., 1947, Charles C. Thomas, p. 72.
- Johnson, J.: The Surgery of Congenital Heart Disease, *Surg. Clin North America* 31: 1811-1820, Dec., 1951.
- Vineberg, Arthur: Development of an Anastomosis Between the Coronary Vessels and Transplanted Internal Mammary Artery, *Canad. M. A. J.* 55: 117, 1946.
- Vineberg, Arthur: Treatment of Coronary Artery Insufficiency by Implantation of the Internal Mammary Into the Left Ventricular Myocardium, *J. Thoracic Surg.* 23: 42-54, 1952.

CHAPTER XVI

SURGICAL TREATMENT OF ESSENTIAL HYPERTENSION

ARTHUR M. VINEBERG, M.D.

Essential hypertension is the name given to a disease in which the blood pressure is elevated without apparent cause. Persistently elevated blood pressures are not normal and cannot be disregarded. In general, it can be stated that at present there is no satisfactory medical treatment for this disease.

Historical.—The history of the surgical treatment of essential hypertension dates back to 1923 when it was first suggested by Kraus to Brünig. These authors suggested that hypertension was caused by spasm of the splanchnic arterioles, and that this spasm might be relieved by section of the splanchnic nerves. Since that time various types of operative procedures have been developed, the most recent of which is the Smithwick operation. In this operation the sympathetic ganglia are removed from the 8th dorsal to the 1st lumbar ganglia along with the splanchnic nerves bilaterally.

Physiological Considerations

Before proceeding to a physiological study of essential hypertension, an examination of the factors controlling normal systemic blood pressure may be of value. The control of the systemic blood pressure is maintained by an extremely complex mechanism. It is dependent upon the integrated function of three main systems as follows:

1. Cardiovascular:

- (a) *Heart:* Alterations in rate or stroke volume are reflected in the blood pressure.
- (b) *Circulating blood volume:* Increase tends to raise blood pressure.
- (c) *Arteriolar tone:* Spasm raises blood pressure.

2. Hormonal Mechanism:

Sympathetico-adrenal syndrome, Cushing's syndrome, etc.

3. Autonomic Nervous System:

Balance is normally maintained by antagonism between parasympathetic and sympathetic:

- (a) Central nervous system control.
- (b) Reflex centers, normally inhibitory, such as
 - (i) carotid sinus mechanism.
 - (ii) depressor mechanisms in the aortic arch.

A disturbance which causes imbalance in any of the aforementioned mechanisms may result in hypertension. Unfortunately, in cases of human essential hypertension there is no definite evidence to show which, if any, of these normal mechanisms for maintaining blood pressure is disturbed.

Physiopathology and Pathogenesis

The etiology of essential hypertension appears to be unknown. It is considered by physiologists and cardiologists that the diastolic blood pressure is a product of peripheral resistance. When the peripheral resistance is increased, there is a corresponding increase in the diastolic pressure. It has been suggested that in cases of essential hypertension the peripheral resistance is increased, due to an exaggerated vasomotor response to a stimulus. Increased peripheral resistance may then be due to:

- (a) autonomic nervous impulses from vasomotor centers;
- (b) renal arteriolar spasm;
- (c) hormonal mediation, i.e., anterior pituitary, adrenal, kidney;
- (d) intrinsic arteriolar myogenic tone mechanisms.

Many clinical and experimental facts have been published, a consideration of which has led to various theories concerning the etiology of essential hypertension.

(a) Neurogenic Theory of Hypertension.

—Supporters of the neurogenic theory of hypertension suggest that essential hypertension results from sympathetic overaction with excessive sympathetic outflow from a cerebral center. Experimentally, neurogenic hypertension can be produced by injecting fluid under pressure into the cisterna magna, or it can be developed by carotid sinus denervation and section of the aortic depressor nerves. In the latter condition, the afferent impulses to the circulatory center in the medulla are interrupted—this allows unrestrained activity of vasoconstrictor, cardio-accelerator and secretory centers. Hypertension lasting three to four years and reaching 250 mm. of mercury has thus been produced. The hypertension which results appears to be due to peripheral vasoconstriction and, experimentally, has been relieved by sympathectomy.

There is some clinical evidence to support the neurogenic thesis of essential hypertension with which all physicians are familiar. The sudden transient elevations of blood pressure which occur in patients on their first examination in a doctor's office are considered to be a normal response to an emotional stimulus. This response becomes greatly exaggerated in the hypertensive patient.

Further, psychiatrists claim to be able to cure certain cases of essential hypertension when the underlying psychiatric disturbances have been removed.

(b) Renal Hypertension.—The kidney as a factor in essential hypertension has been suspected for some time. However, it has never been clear as to which was the cause and which the effect. Many cases of hypertension have shown no renal disease, and conversely, many cases of chronic nephritis have failed to develop hypertension. There

is much evidence to indicate that the renal arteriolar changes found in advanced cases of essential hypertension are secondary to the disease and are not present in its early stages. Castleman and Smithwick have suggested that the arteriosclerotic changes which are found in renal vessels of patients suffering from essential hypertension are secondary to the hypertension rather than its primary cause.

In 1940, Goldblatt and his co-workers described experiments in which hypertension was produced after interference with the renal blood flow. The renal circulation was diminished by means of a metal clamp placed upon one or both renal arteries, or upon the abdominal aorta. The hypertension thus produced occurred without impairment of kidney function and disappeared upon removal of one or both clamps. Renal ischemia results in the production of a vaso-spastic substance known as *renin*. It is this substance which is said to cause the hypertension produced by clamping the renal vessels. The work of Goldblatt and his co-workers led to an intensive search for unilateral renal disease in cases of essential hypertension. It was suggested that pyelonephritis, arteriolar sclerosis, aneurysm of the main artery of the kidney, compression or deformity of one main renal artery, etc., capable of producing a unilateral disturbance of intrarenal hemodynamics, might be the cause of essential hypertension in certain cases. There have been a few cases reported in which this appeared to be true, and in which the blood pressure returned to normal after removal of the diseased kidney. The presence of unilateral renal lesions causing renal ischemia has been demonstrated comparatively rarely in cases of hypertension. Experimental hypertension produced by the Goldblatt clamp is not relieved by sympathectomy.

Spasm of renal arteriolar vessels does occur. It has been shown by Trueta, et al, that ischemia of the renal cortex can co-exist

in a kidney with a good medullary blood supply. Renin, or other pressor substances, may be liberated under such conditions. It is thus possible that renal arteriolar spasm may be responsible for essential hypertension. This may occur in much the same way as does peripheral arteriolar spasm in Raynaud's disease.

Sympathectomy is of value in Raynaud's disease in relieving arteriolar spasm, and should likewise be of value in relieving renal arteriolar spasm. There is a possibility that continued neurogenic hypertension, with its associated continuous renal arteriolar spasm and renal ischemia, may result in secondary arteriosclerotic change in the renal vessels.

(c) **Hormonal Hypertension.**—For many years the adrenal glands have been suspected of playing a part in the development of essential hypertension, and there is much evidence to support this conception. Thus, adrenal tumors (i.e., pheochromocytomas) arising in the adrenal medulla are responsible for a type of hypertension which disappears after the tumor has been removed. There is also experimental evidence to show that hypertension due to renin formation cannot occur in the absence of the adrenal cortex.

More recently, Heinbecker has suggested that essential hypertension is due to hormonal influences. It is suggested that the adrenal is stimulated indirectly through the pituitary which causes a constriction of afferent renal arterioles (glomerular) with the production of renin and resultant hypertension.

(d) **Intrinsic Arteriolar Myogenic Tone Mechanism.**—One of the most important factors in the maintenance of blood pressure is that of the resistance to blood flow offered by the arterioles throughout the body. Normally, the vast arteriolar bed is responsive to nervous and hormonal stimuli. Spasm of the arterioles increases peripheral resistance and thus raises the blood pressure. Conversely, dilatation of the arterioles decreases

the peripheral resistance with a resultant lowering of blood pressure.

It has been suggested that the increased tone of the arteriolar muscle causes essential hypertension. The increase of arteriolar tone is intrinsic and is therefore little influenced by extraneous stimuli, whether of neurogenic or of hormonal origin.

Anatomical Considerations

The adrenal glands, the renal and splanchnic arterioles, separately or together, may be involved in the production of essential hypertension. Their sympathetic nerve supply commences in the cerebrum, passes down the spinal cord and reaches the peripheral ganglia through the lateral horn cells.

It is important to note that the great splanchnic nerve, which normally originates from the 5th to the 9th dorsal ganglion, may arise from as high as the 2nd dorsal ganglion

RATIONALE UNDERLYING SYMPATHECTOMY FOR ESSENTIAL HYPERTENSION

It has been shown experimentally, that sympathectomy relieves the hypertension of neurogenic origin. If essential hypertension is caused by renal arteriolar spasm or by adrenocortical hormone activity, directly or indirectly, then interruption of the nerve supply to the adrenal gland and adrenal vessels should be of value. Particularly is this true of the adrenal gland which is supplied almost entirely by the great splanchnic nerve and which shows atrophic degeneration of the anterior half of its medulla following section of the great splanchnic nerve. If the increased diastolic pressure is caused by increased arteriolar spasm through splanchnic stimulation, then sympathectomy should relieve the spasm. No effect, theoretically, can be expected from sympathectomy if irreversible arteriolar changes have occurred in the kidney or the peripheral vascular bed.

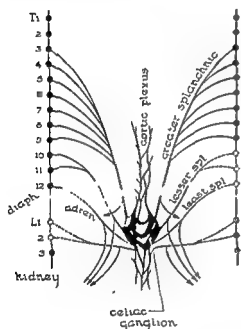
SURGICAL OBJECTIVES IN THE TREATMENT OF ESSENTIAL HYPERTENSION

- 1 To interrupt the fibers carrying vaso-spastic and other impulses from the brain to the following areas or organs:

- (a) adrenal glands
- (b) renal vessels
- (c) splanchnic vascular bed
- (d) vessels of the lower limbs

of a resection of the splanchnic nerves, a portion of the celiac ganglia and the upper lumbar trunks to include the 1st and 2nd lumbar ganglia. By the subdiaphragmatic approach Adson attempted to denervate the adrenal glands and the splanchnic and renal arteriolar beds.

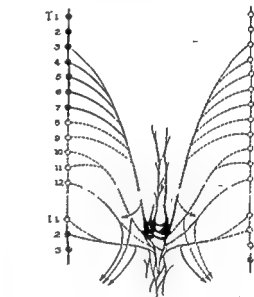
2. **Supradiaphragmatic Operation.**—First performed by Peet in 1933. The 11th and 12th dorsal ganglia and the greater, lesser,



subdiaphragmatic
(Adson)



supradiaphragmatic
(Peet)



thoraco-lumbar
(Smithwick)



radical thoraco-lumbar
(Grimson)

(——— nerves resected)

Fig 154—Composite diagram showing four types of sympathectomy used in the treatment of essential hypertension

- 2 To prevent nerve regeneration.
3. Exposure should be such as to allow for exploration of the adrenal gland and to permit of renal biopsy.
4. To have as low a mortality and morbidity as is consistent with the desired effect
- 5 The operation theoretically should be pre-ganglionic rather than post-ganglionic in order to prevent sensitization to adrenalin.

SELECTION OF OPERATION

1. **Subdiaphragmatic Operation.**—First carried out by Adson in 1936 and consisting

and least splanchnic nerves were bilaterally removed through a supradiaphragmatic exposure

3 **Thoracolumbar Operation.**—In 1940 Smithwick removed the sympathetic chain from the 8th dorsal ganglion to the 1st or 2nd lumbar ganglion along with the greater, lesser and least splanchnic nerves, through a combined supra- and infradiaphragmatic approach. The diaphragm is separated from the 1st lumbar vertebra which permits of a better exposure of the sympathetic chain

and ganglia. This operation is performed bilaterally.

The type of operation most likely to satisfy the surgical objective in the treatment of essential hypertension is the thoracolumbar operation.

Mitchell, on anatomical grounds, suggests that the optimum denervation of the adrenal glands, splanchnic bed, and renal vessels can be obtained by the removal of the sympathetic ganglia from D 4 down to L 3 along with the greater, lesser and least splanchnic nerves. This procedure also denervates the vessels in the lower limb. The sympathectomy is preganglionic in type and has been clinically carried out by numerous surgeons (Hinton, Poppen and others). In the young male the operation should not extend beyond L 1 ganglion—removal of L 2 ganglion results in the loss of ejaculation with resultant sterility.

INVESTIGATION OF CASES OF ESSENTIAL HYPERTENSION FOR SYMPATHECTOMY

The diagnosis of essential hypertension can be made only after a careful search has been conducted to exclude diseases of the endocrine, central nervous, and genitourinary systems. At present there are no definite tests which can foretell whether or not a patient will obtain a good result from sympathectomy. However, it is possible, on the basis of clinical experience, to know beforehand which cases will definitely be made worse or be uninfluenced by sympathectomy. In order to determine those cases which may do badly or die, certain information concerning the condition of the arteriolar system in the brain, the eyes, the cardiovascular system and the kidneys must be obtained.

Cerebral

A carefully taken history may reveal symptoms of irritability, aphasia, transitory loss of consciousness and groping for words. Such symptoms indicate cerebral anemia with a secondary cerebral degeneration.

Cardiovascular System

Heart.—Investigation for cardiac enlargement, disease of coronary arteries, and valvular lesions requires that the heart be examined by a competent cardiologist and that an electrocardiograph and a six-foot x-ray film plate of the chest be taken. The importance of blood pressure levels and variations has been stressed by some investigators. Others, however, conclude that the various blood pressure studies and tests are of very little prognostic value.

Blood Pressure Tests.—

Home Blood Pressure Measurements.—A relative or friend of the patient is instructed how to take the blood pressure of the patient at different intervals throughout the day. The patient is given a book in which the home blood pressure readings are recorded over several months. Blood pressure readings obtained in this way, it is felt, reflect more accurately the average blood pressure of the patient than can be obtained by any other method.

Cold Pressure Test (Hines).—After the patient has been at rest in a quiet room for at least half an hour, blood pressure readings are taken every minute for five minutes, in the lying, sitting, and standing positions. These readings are repeated during and after the right hand has been immersed in ice water for a period of 60 seconds. Five readings are taken at minute intervals with the patient at rest. The right hand is then placed in ice water, the blood pressure reading repeated at 30 and 60 seconds and then at minute intervals for another 5 minutes. This cold pressure test is repeated in the sitting and standing positions. With each blood pressure determination the pulse rate is recorded. By means of this test it is considered by Smithwick and others that it is possible to estimate the peak load to which the cardiovascular system is subjected under periods of stress and strain. The blood pressure response rise to a cold stimulus in certain cases may be very marked. Following sympathectomy, in many instances it is possible to demonstrate

a reduction in the blood pressure response to the same stimulus

Sedation Test (Sodium Amytal).—Some writers consider that this test in 80% of cases shows what blood pressure change may be expected following sympathectomy. Sodium Amytal, 3 gr., is given to the patient at 9:00, 10:00 and 11:00 P.M., respectively, and the blood pressure and pulse recorded every hour throughout the night along with a record as to whether the patient was asleep or not at the time of taking the blood pressure.

There are other tests which are employed in an attempt to judge as to which case will obtain a good result and which one will not. These comprise the exercise, tetraethylammonium-chloride, spinal anesthesia tests, and others, most of which have not proved to be very satisfactory.

Renal.—The extent of renal damage is very difficult to determine preoperatively. The following tests of renal function appear to be of value:

1. Blood chemistry: Nonprotein nitrogen, creatinine and urea
2. Mosenthal and P.S.P
3. Uroselectan and retrograde pyelogram.

The urea clearance and insulin tests are not generally used.

Eyes.—Examination of the fundi should be carried out by a competent ophthalmologist in every patient before and after sympathectomy. Fields of vision and visual acuity should be recorded.

SELECTION OF CASES OF ESSENTIAL HYPERTENSION FOR SURGERY

At present there is no accurate means of selecting cases of essential hypertension for sympathectomy. There are many important factors to be considered, and a favorable prognosis cannot be given in any one case.

Information concerning the "age" of the arterial tree is vitally important. Particularly is this true of the vessels of the brain, heart, and kidneys. The age of the patient gives little information as to the condition

of the patient's arteries. Frequently, the arterial system of a young individual displays a degree of arteriolar degeneration commonly found in the aged.

In general, patients under 40 years of age, without complications, obtain the best results from sympathectomy. However, many patients between 50 and 59 years of age have been operated upon with excellent end results. The duration of the disease seems to play no important part in the prognosis. This is understandable as some patients may have an elevated blood pressure for many years without obvious damage to the arteriolar system. Others may have hypertension for only a few months and yet suffer irreparable damage to their arteriolar systems. In the latter event, sympathectomy for the relief of splanchnic or renal arteriolar spasm can be of little use.

The arteriolar system in cases of essential hypertension (as indicated by blood pressure variation under Sodium Amytal, cold pressure and exercise tests) must be sufficiently resilient to allow for a reversal of the disease process. When the blood pressure (systolic and diastolic) returns to normal under Sodium Amytal, or with rest, the prognosis is said to be good.

Perhaps the greatest contraindication to surgery is the involvement of one or more major organs, other than the eyes. Thus, patients showing signs of encephalopathy, coronary thrombosis, and angina pectoris are poor risks. Likewise, are cases of renal failure, as evidenced by an elevated nonprotein nitrogen and a reduction of intravenous phenolsulphonphthalein output to below 15% in the first fifteen minutes. On the other hand, hemiplegia, compensated congestive heart failure, and positive urinary findings, such as red blood cells, casts and albumin, do not alone constitute contraindications to surgery.

It is doubtful whether the life expectancy of patients with renal failure, coronary thrombosis, and advanced encephalopathy can be prolonged by the lowering of blood pressure. There is also some question

whether patients suffering from hypertension which is symptomless should be subjected to surgery. In effect, surgery should only be performed upon patients who are to a greater or lesser extent incapacitated by their hypertension.

RESULTS OF SURGICAL AND MEDICAL TREATMENT OF ESSENTIAL HYPERTENSION

The results of treatment of essential hypertension are extremely difficult to evaluate. Comparison between various groups of patients contributes to the difficulty because there are many stages of the disease which may occur at any age in both sexes. Some cases may show marked blood pressure reduction without alleviation of headache, dizziness, and other symptoms. The reverse may be true. Comparable groups of well-studied and well-classified cases must be followed with and without treatment from 5 to 10 years. This will be necessary before definite conclusions can be drawn concerning the value of any treatment, whether it be medical or surgical.

There are many reports in the literature concerning the value of both medical and surgical treatment. In one of the recent reports a comparison between medically and surgically treated cases of essential hypertension is made. In this report 100 cases were carefully followed for a period of 3 years. Both the control (medically treated) and the surgically treated cases were severe, with many complications at the start of the observation period. In the control group of patients there were 61 complications such as ventricular failure, cerebral accidents, angina pectoris, and myocardial infarction. In the sympathectomy group there were 73 complications of the same type. After 3 years in the control group of patients treated medically there was only one patient in good condition, 4 were fair or improved, 4 were unchanged, and 41 were worse or dead. In the sympathectomy group of patients there were 11 excellent results; 11 with fair and

definite improvement; 5 showed no change; 11 were worse; 12 were dead. There is agreement among all authors that 12 to 20% of postsympathectomy patients will be markedly improved at the end of 5 to 10 years. Such patients should be symptom free and have normal blood pressures.

REFERENCES

- Adson, A. W., and Brown, G. E.: *J. A. M. A.* 102: 1115, 1934.
 Adson, A. W., Lundy, J. S., and Allen, E. V.: *Proc. Staff Meet., Mayo Clin.* 11: 401, 1936.
 Alexander, W. F., Kuntz, A., Henderson, N. P., and Ehrlich, E.: *J. Internat. Coll. Surgeons* 12: 115, 1949.
 Bacq, Z. M., Brouha, L., and Heymans, C.: *Arch. internat. de pharmacodyn. et de therap.* 48: 429, 1934.
 Blackford, J. M., and Wilkinson, J. N.: *Ann. Int. Med.* 6: 54, 1932.
 Bruning, F.: *Klin. Wchnschr.* 2: 777, 1923.
 Cushing, W.: *J. A. M. A.* 101: 1115, 1934.
 Evelyn, K. A., Alexander, F., and Cooper, S. R.: *J. A. M. A.* 140: 592, 1949.
 Goldblatt, H.: *J. Exper. Med.* 65: 671, 1937.
 Goldblatt, H.: *Introductory Lecture on the Production and Pathogenesis of Experimental Hypertension*, N. Y. Acad. Sc. Spec. Pub. 3: 4-31, 1946.
 Grimson, K. S.: *Ann. Surg.* 114: 753, 1941.
 Grimson, K. S., Orgain, E. S., Anderson, B., Broome, R. A., and Longino, F. H.: *Ann. Surg.* 129: 850, 1949.
 Heinbecker, P.: *Ann. Surg.* 126: 535, 1947.
 Hinton, J. W., and Lord, J. W., Jr.: *New York State J. Med.* 46: 1015, 1946.
 Insurance Editorial: *J. A. M. A.* 117: 49, 1941.
 Keith, N. M., Wagener, H. P., and Barker, N. W.: *Am. J. M. Sc.* 197: 332, 1939.
 Keith, M. A., Woolf, B., and Gilchrist, A. R.: *Brit. Heart J.* 12: 287, 1949.
 Mitchell, G. A. G.: *Edinburgh M. J.* 54: 545, 1947.
 Page, I. H., and Corcoran, A. C.: *Arterial Hypertension Its Diagnosis and Treatment*, Chicago, 1945, The Year Book Publishers, Inc.
 Peet, M. M.: *California Med.* 5: 58, 1935.
 Poppen, J. L.: *Surg., Gynec. & Obst.* 84: 1117, 1947.
 Rowntree, L. G., and Adson, A. W.: *J. A. M. A.* 85: 959, 1925.
 Smithwick, R. H.: *Surgery* 7: 1, 1940.
 Smithwick, Reginald: *Surgical Measures in Hypertension*, Springfield, Ill., 1951, Charles C. Thomas.
 Trueta, J., Barclay, A. E., and Daniel, P. M.: *Lancet* 2: 237, 1945.
 White, Paul D., Dumond, E. Grey, and Williams, Armistead: *Follow-up Study of One Hundred Private Hypertensive Patients With Cardiovascular Complications*, *J. A. M. A.* 143: pp 1311-1317, 1950.

CHAPTER XVII

ESOPHAGUS

ARTHUR M. VINEBERG, M.D.

INTRODUCTION

Many factors make surgery of the esophagus technically difficult. Some of these are related to anatomical peculiarities. Others have to do with the ordinary problems of intrathoracic surgery, such as control of the open thorax and the maintenance of lung expansion sufficient to ensure adequate oxygenation of the blood. Because of great advances in the field of anesthesiology, the thorax may now be opened with almost as little risk as the abdomen. However, it was as recently as 1938, that Phemister and Adams were the first to perform a successful one-stage resection of the esophagus and the cardiac portion of the stomach.

APPLIED ANATOMY AND PHYSIOLOGY

The esophagus is a muscular canal which extends from the pharynx to the stomach. Its wall is composed of three layers: an inner mucous membrane, a submucous layer and an outer muscular layer. Unlike other parts of the gastrointestinal canal, the esophagus has no serosal covering. The mucous membrane is of the squamous cell type except for the most distal portion where the cells become columnar in character.

The main function of the esophagus is to convey fluid and food from the pharynx to the stomach. Food is propelled down the esophagus by peristaltic waves, but fluids descend mainly by gravity. Before food or fluid can enter the stomach, the cardiac sphincter must be relaxed. The mechanism which opens the sphincter is obscure, though its control is thought to be through the autonomic nervous system.

Normally, the esophageal lumen is narrowed at three points:

- (a) the level of the cricoid cartilage
- (b) where it is crossed by the left bronchus
- (c) where it pierces the diaphragm

The esophagus is divided into cervical, thoracic, and abdominal portions. The *cervical portion* begins at the lower border of the cricoid cartilage, opposite the sixth cervical vertebra. Anteriorly, lie the trachea and the thyroid gland while posteriorly are the vertebral column and the longus colli muscle. The common carotid arteries and part of the lateral thyroid lobes lie on either side of it. Between it and the trachea, the right and left recurrent nerves ascend. The thoracic duct lies to its left side.

The *thoracic portion* begins at the thoracic inlet, and ends where the esophagus penetrates the diaphragm. For surgical reasons, it is divided into two segments: a *midthoracic segment*, comprising supra- and infra-aortic portions, and a *lower thoracic segment* from the root of the lungs to the diaphragm. After entering the thorax, the esophagus rests upon the vertebral column, the longus colli muscle, the right intercostal arteries, the thoracic duct, the hemiazygos and accessory hemiazygos veins. In front are the trachea, the left main bronchus, pericardium and diaphragm. On the left side, in the supra-aortic part, lie the left subclavian artery, thoracic duct and the left pleura, with the left recurrent laryngeal nerve between the trachea and the esophagus. In the infra-aortic portion, the thoracic duct lies posteriorly, with the descending aorta and pleura on its left. On the right side are

pleura and the azygos vein which latter is partly covered by the esophagus. The vagus nerves descend in contact with the esophagus below the roots of the lungs, the right lying behind and the left in front.

The relationship of the thoracic duct to the esophagus is of great importance since this structure may be easily damaged dur-

The abdominal portion of the esophagus lies in the esophageal groove on the posterior surface of the left lobe of the liver. It is covered by peritoneum in front and on its left side.

Blood Supply.—The blood supply of the esophagus, which is much richer than was previously supposed, is segmental in distribu-

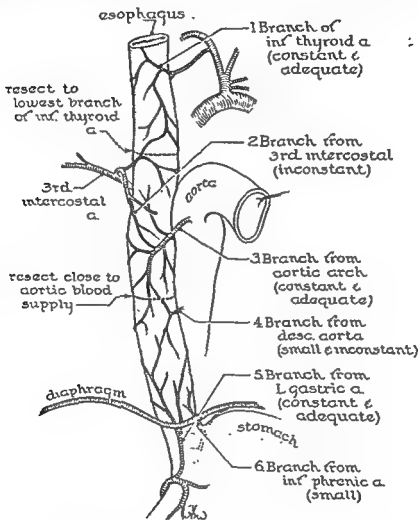


Fig 155—Segmental arterial blood supply of the esophagus.

ing esophageal manipulation. In the infra-aortic segment the thoracic duct lies posterior to and on the right of the esophagus. As it ascends it passes slightly to the left, lying closer to the esophagus. In the supra-aortic segment the duct continues forward and, about midway in the superior mediastinum, ascends anteriorly and to the left until it enters the neck and terminates in the left subclavian vein.

The vessels, for the most part, run in a longitudinal direction. From above downward, the most important individual arteries are the following:

1. Inferior thyroid, supplying the cervical and supra-aortic segments.
2. Branches from the inferior surface of the aortic arch.
3. Branches of the bronchial arteries.

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Constriction of the esophagus by an anomalous vessel is an exceedingly rare condition in which, because of a persistent embryological pattern, there is a double aorta which forms a ring about the trachea and the esophagus. One of the two vessels making up the aortic ring is always much larger than the other. The smaller vessel usually lies anterior to the trachea and frequently is no more than a fibrous constricting band.

Treatment consists of freeing the constricting ring by division of the lesser vessel.

ected and lead to abscess formation. When necessary, such a cyst may be excised and the esophageal wall reconstructed.

BENIGN ESOPHAGEAL OBSTRUCTION

Benign esophageal obstruction may be caused by foreign bodies, or by acute or chronic inflammatory states resulting from mechanical, thermal or chemical (strong acid or alkaline) irritation. There are cases

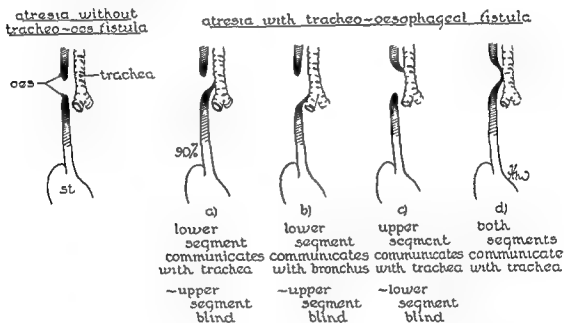


Fig 156—Congenital atresias of the esophagus without and with tracheoesophageal fistulas

Dysphagia Lusoria.—An aberrant right subclavian artery may cause partial obstruction of the esophagus. The anomalous vessel arises from the posterior surface of the aortic arch on the left side and passes behind the esophagus. The pressure produced by such a vessel interferes seriously with deglutition. Double ligation of the artery close to its origin immediately relieves the dysphagia.

Congenital cysts within the wall of the esophagus may be bronchogenic in origin or composed of elements of the esophagus itself. They tend to increase in size and cause obstruction. They may become in-

fect of esophagitis of obscure etiology in which regurgitation of acid gastric secretion may be a contributing factor. Duodenal and esophageal ulceration, short congenital esophagus and hiatus hernia are often associated with this condition. It should be noted that esophagitis with ulceration may result from repeated or prolonged gastrointestinal intubation.

Ulcers of the esophagus may be acute or chronic. The chronic ulcer is usually found in the distal end of the esophagus and has many of the characteristics of a peptic ulcer. Healing ulcers may cause benign stricture of the esophagus.

4. Branches of the thoracic portion of the descending aorta
5. Inferior phrenic artery.
6. Left gastric artery.
7. Superior suprarenal arteries.

The veins from the lower end of the esophagus open into the left gastric vein which is a tributary of the portal vein. Otherwise, esophageal veins generally follow the course of their associated arteries.

Lymphatics.—From above downward, the lymphatic drainage of the esophagus is received by supraclavicular, superior mediastinal, hilar and lower mediastinal nodes. Below the diaphragm, esophageal lymph drains into the cardio-esophageal, left gastric and lesser curvature nodes

EMBRYOLOGY

In early fetal life the esophagus and trachea are one tube, which is divided into two, between the fourth and twelfth weeks, by an ingrowth of mesoderm. Epithelial cells lining the esophagus rapidly proliferate and occlude the lumen. The lumen is subsequently re-established by vacuoles which coalesce. Failure of ingrowing mesoderm to separate completely the esophagus and trachea results in tracheo-esophageal fistula. Atresia occurs when the solid core of esophageal cells does not vacuolize, or when vacuoles develop but fail to coalesce.

CONGENITAL ANOMALIES

Anomalies of the esophagus are encountered about once in two thousand births. Unless such conditions are recognized and treated within a very short time after birth, the child will not survive.

Agenesis of the thoracic esophagus is exceedingly rare. Congenital atresia and tracheo-esophageal fistula are better known surgical problems. Atresia of the esophagus may be present without a tracheal fistula. In such cases there may be complete separation of the esophagus into upper and lower segments. Occasionally, the segments are joined together by a fibrous cord. The point

of atresia may consist of a relatively thin diaphragm obstructing an otherwise normal lumen. Atresia of the esophagus complicated by tracheo-esophageal fistula has been reported to occur in four different ways. In 90 per cent of such cases the upper esophageal segment is blind and the lower segment forms a fistulous tract with the trachea.

Symptoms.—The symptomatology varies according to the type of congenital anomaly present. When the upper segment terminates blindly, food is immediately regurgitated, and there is excessive salivation. If the upper segment communicates with the trachea or a bronchus, food is aspirated into the respiratory tree. Dyspnea and cyanosis are the most prominent symptoms in such cases. When the distal segment communicates with the trachea, air enters the stomach and intestines, and asphyxia results from the regurgitation of intestinal contents.

Diagnosis.—Cyanosis, regurgitation, and excessive salivation are the cardinal physical signs. When the lower esophageal segment communicates with the trachea, a flat film of the abdomen will show distention of the stomach with air. Inability to pass a catheter down the esophagus will indicate the lower end of the blind upper segment. If the position of a catheter tip is in doubt, injection through it of a small amount of radio-opaque oil may be helpful.

Treatment.—Early diagnosis and treatment of tracheo-esophageal fistula are imperative. Once the condition has been recognized, the immediate problem is the prevention of food and intestinal secretions from entering the lungs. Preoperatively, this is accomplished in part by posture and continuous suction of the proximal segment of the esophagus. If the nature of the lesion permits, the fistula should be closed and the esophageal segments joined end to end. Where primary anastomosis is not possible, the fistula is closed and cervical esophagotomy and gastrostomy are performed. Establishment of esophageal continuity may be attempted at a later date.

Extravasation of radio-opaque oil introduced into the esophagus further confirms the rupture. Aspiration of the pleural cavity yields fluid characteristic of gastric or esophageal contents.

Treatment.—Control of shock, decompression and drainage of the tension pneumothorax and replacement of oral feedings by parenteral fluids are immediate requirements. Chemotherapy is instituted. Thoracotomy is performed with closure of the esophageal tear. The mediastinum and pleura are cleansed and drained. A nasal catheter is introduced to prevent postoperative distention of the stomach.

PERFORATION OF THE ESOPHAGUS

Perforation of the esophagus occurs fairly frequently. It is caused by trauma, inflammation, or neoplasia.

Traumatic perforation results from external violence or from internal injury. The passage of a bougie or esophagoscope may cause perforation, especially through an unsuspected esophageal diverticulum. Foreign bodies, in particular fragments of bone and safety pins, sometimes penetrate the esophageal wall. External violence may lead to rupture of the esophagus in association with damage to other organs.

Inflammatory perforation may follow the ingestion of corrosive liquids or the penetration of a chronic ulcer.

Neoplastic perforation does not usually occur until the lesion has encroached upon the lumen sufficiently to produce symptoms of esophageal obstruction.

Symptoms.—In general, the symptoms of perforation of the thoracic esophagus are the same as those recorded for spontaneous rupture. Their severity depends upon the size of the perforation and the rapidity with which it develops. Perforation of the cervical esophagus may or may not be accompanied by hydropneumothorax.

Treatment is the same as that of spontaneous rupture of the esophagus.

IDIOPATHIC DILATATION OF THE ESOPHAGUS

(SYNONYMS: CARDIOSPASM, ACHALASIA)

Early theories suggested that in cardiospasm, esophageal dilatation was produced by failure of the cardiac sphincter to open reflexly during the act of swallowing. The term *achalasia*, meaning lack of relaxation, was introduced subsequently. Absence of hypertrophied muscle fibers in the narrowed portion of the esophagus led others to assume that there had occurred progressive degeneration of Auerbach's plexus in the region of the epicardia. Whatever the cause, cardiospasm or idiopathic dilatation of the esophagus accounts for dysphagia in approximately 20 per cent of cases. It is commoner in the female than in the male. Although cardiospasm has been recorded in infancy, most cases appearing for treatment are in the third or fourth decade of life. There seems to be a definite psychogenic factor.

Pathological Features.—There is a narrowed, thin, pale area, 3 to 6 cm in length just proximal to the cardia; the muscular coats are to some degree replaced by fibrous tissue, and degeneration of myenteric ganglia is evident.

The radiological deformity is at first a funnel-shaped dilatation which, in the later stages, becomes flask-shaped, and ultimately assumes a sigmoid form.

Symptoms.—Patients with cardiospasm can feel the esophagus contract when they are excited. The first swallows of food pass easily, but there soon follows a sensation of fullness behind the lower portion of the sternum. Symptoms of dysphagia are aggravated by rapid eating, roughage, and hot or cold fluids. As the disease progresses, regurgitation occurs, and weight is lost. Nocturnal regurgitation may result in aspiration pneumonia or pulmonary abscess.

Diagnosis.—X-ray examination and esophagoscopy are confirmatory. The latter is particularly helpful in distinguishing between cardiospasm and esophageal carci-

Symptoms.—These depend upon the severity of the esophagitis and the degree of stricture. Intense thermal or caustic irritation causes extreme dysphagia of rapid onset, associated with varying degrees of shock. The presence of foreign body obstruction may be obvious because of the history, or unsuspected until revealed by x-ray or esophagoscopy. Dilatation of the lower third of the esophagus is frequently associated with epigastric and substernal pain. Hematemesis may result from the presence of ulceration or of an acute diffuse esophagitis.

In chronic esophagitis, complicated by stricture formation, the onset of symptoms is gradual. Dysphagia increases until regurgitation occurs, and finally, even fluids fail to pass the obstruction.

Diagnosis.—A history of swallowing a foreign body or some chemical or thermal irritant is usually obtained. In cases of chronic obstruction, lacking a clear-cut history of local injury, the problem is to differentiate between benign and malignant strictures. In many instances, x-rays are inconclusive and, in any event, should be followed by esophagoscopy with biopsy where indicated.

Treatment.—This depends upon the cause of the esophagitis and the degree to which the lesion has progressed. When caustics have been swallowed, usually with suicidal intent, gastrostomy is frequently required following emergency measures to neutralize and remove excess of the irritant. Foreign bodies, when not vomited, or passed further into the gastrointestinal tract, are removed by the esophagoscope or, if this is impossible, by local esophagotomy. In subacute and chronic esophagitis without stricture, a bland diet, atropine and antacids are helpful. When a stricture is present, it may yield to mechanical dilatation with bougies. If it does not, or if symptoms recur following repeated dilatations, resection of the lesion becomes necessary.

SPONTANEOUS RUPTURE OF THE ESOPHAGUS

Spontaneous rupture of the esophagus is an extremely rare condition which occurs four times more frequently in males than in females. In certain cases, the cause is some pre-existing weakness of the esophageal wall, which gives way when intracophageal pressure is suddenly increased. This usually occurs in the lower third on the left posterolateral wall, and is followed by rupture into the left pleural space. It may, however, occur through the left anterolateral wall. The mortality rate which in the past was 100 per cent has been reduced in recent years to approximately 30 per cent by early diagnosis and prompt surgical intervention.

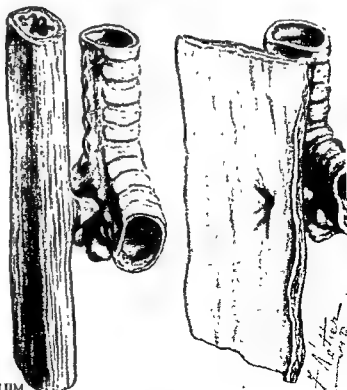
Diagnosis.—The patient is commonly a previously healthy male, robust, a heavy eater, and possibly an alcoholic, who complains of excruciating pain immediately following a bout of vomiting, which may have been either spontaneous or induced. The pain is epigastric, radiating to the left side of the chest, and to the back. Dyspnea, cyanosis, and shock develop rapidly. Hematemesis is rarely copious. Vomiting generally ceases after the onset of pain and is followed by intense thirst. Some patients present a history of previous ulcer, esophagitis or esophageal stricture. Physical examination reveals severe shock and a unilateral (usually left) or bilateral hydropneumothorax. Emphysema is present at the base of the neck in 60 per cent of cases. Perforated peptic ulcer, acute pancreatitis, mesenteric thrombosis, dissecting aneurysm of the aorta, coronary thrombosis, pulmonary embolism, and spontaneous pneumothorax must be excluded.

The presence of hydropneumothorax associated with shock and vomiting is almost pathognomonic. At an early stage, x-rays reveal mediastinal emphysema, which ascends rapidly into the cervical region. They may also demonstrate fluid levels in the mediastinum and in the pneumothorax.

PULSION DIVERTICULUM



TRACTION DIVERTICULUM



noma In advanced cases, aspiration by catheter will recover putrefying food products.

Treatment is divided into medical and surgical phases. Medically, a bland diet, antispasmodics, and psychotherapy are worthy of trial. If the disease progresses, dilatation by means of a mercury-loaded bougie, or by a hydrostatic bag, is said to relieve a large proportion of cases.

If, after conservative therapy, symptoms persist and lead to malnutrition, surgical intervention becomes necessary. The operation may consist of resection and re-anastomosis; longitudinal incision of the muscular coats of the narrowed segment, lateral esophagogastric anastomosis, or various forms of cardioplasty.

ESOPHAGEAL DIVERTICULA

Pulsion and traction diverticula occur in the esophagus.

Pulsion diverticulum is a herniation of mucosa and submucosa through the wall of the esophagus. Such lesions are divided into three types, according to their extent

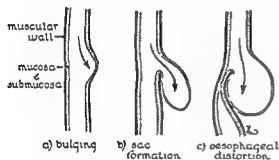


Fig 157 —Stages of esophageal diverticula.

of development: stage of bulging, stage of sac formation, and stage of esophageal distortion. Pulsion diverticula occur at the pharyngoesophageal junction and in the lower third of the thoracic esophagus. Pharyngoesophageal diverticula are most common

Etiology.—The pharyngeal constrictor muscles contract upon the bolus of food and

attempt to push it past a zone of constriction which is usually caused by failure of the cricopharyngeus muscle to relax. The result is a bulge in the pharynx above the point of constriction which slowly grows until a diverticulum is formed. The herniation occurs either through the fibers of the inferior pharyngeal constrictor muscle or those of the cricopharyngeus.

Symptoms depend upon the stage of sac development. They are directly referable to the act of swallowing, and may vary from minimal or no discomfort to progressive dysphagia and regurgitation. As the sac enlarges forming a neck, food, air and mucus are retained, causing gurgling noises and regurgitation, particularly when the patient is recumbent. Progressive growth of the sac into the superior mediastinum drags down the lateral esophageal wall and converts the esophageal opening into a slit. In such cases, complete esophageal obstruction may ensue.

Diagnosis.—In addition to a suggestive history, x-rays with barium swallow will demonstrate a sac which communicates with the esophagus. Esophagoscopy, which may be employed for confirmatory purposes, carries with it the danger of perforation through the diverticulum.

Treatment.—When symptoms indicate surgical intervention, the sac may be freed and its fundus sutured to the tissues of the neck at a point superior to its aperture (diverticulopexy). This procedure prevents the diverticulum from filling with food and secretions. When removal of the sac is advisable, a two-stage operation was originally practiced. The diverticulum was freed and marsupialized at the operation as a safeguard against deep cervical and mediastinal infection. It was removed at the second operation, a week or so later. Chemotherapy has lessened the risk of single-stage removal and at the present time this is the operation of choice.

Diverticula in the lower third of the esophagus are comparatively rare. They occur

usually through the right posterior wall, three to four inches from the cardia, and frequently extend into the left thorax. The commonly suggested causes of such lesions are congenital weakness of the esophageal wall, cardiospasm or some other obstacle to deglutition.

Symptoms.—Dysphagia and low sub-sternal pain are the presenting symptoms. Their severity depends upon the size of the sac and the presence of inflammatory changes. There may be choking attacks and regurgitation, particularly upon lying down. Sacs of the third stage are usually associated with progressive esophageal obstruction. The chief complication is ulceration of the diverticulum with perforation into the pleural cavity or mediastinum.

Diagnosis is established by the clinical history and x-ray examination. Many thoracic diverticula are completely asymptomatic, being discovered unexpectedly during x-ray studies of the upper gastrointestinal tract.

If symptoms are marked, excision of the diverticulum must be carried out. The surgical approach is through a left transthoracic incision. In most cases, the lesion may be excised close to the esophageal wall. At times, it is necessary to resect the lower end of the esophagus together with the diverticulum and perform an esophagostomy.

Traction diverticula are caused by cicatricial contraction following inflammation involving the esophageal wall. In such cases the whole wall of the esophagus is pulled out to form the diverticulum. Usually, the inflammatory process originates in the lymph nodes at the hilum of the lung. These diverticula commonly occur in the middle third of the esophagus and rarely produce symptoms.

TUMORS OF THE ESOPHAGUS

The most common obstructive lesion of the esophagus in the adult is a primary carcinoma. Benign tumors such as polyps, fibromas and leiomyomas sometimes cause obstructive symptoms and often can be re-

moved without resection of the esophagus. Fibromas and leiomyomas are intramural and encapsulated, so that they can be dissected out without damage to the mucosal layer.

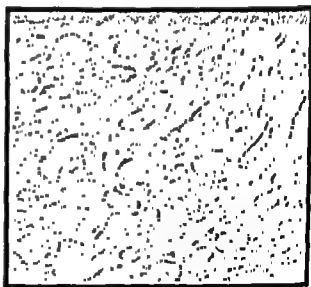
Carcinoma of the esophagus accounts for approximately 4 per cent of deaths from malignant disease. This lesion is classified according to its site of origin; cervical, mid-thoracic (supra-aortic and infra-aortic), lower thoracic and abdominal.

Pathological Features.—Esophageal carcinomas are mostly squamous cell in type, but an adenocarcinoma may arise at any level. Twenty per cent of carcinomas invading the cardiac end of the stomach arise in the lower end of the esophagus and are usually epidermoid in nature.

The *spread* of esophageal carcinoma is either by direct extension through the submucosa and the muscular layers, involving adjacent structures such as the left main bronchus, or by metastases to regional lymphatic nodes. Lymph node involvement occurs early in cancer of the esophagus. Except in the supra-aortic portion, where the cervical glands are involved, the direction of lymphatic spread is downward. In the middle and lower thoracic segments, nodes in relationship to the hilum of the lung, the abdominal esophagus and the left gastric vessels are successively involved.

Symptoms.—Dysphagia is the most constant symptom of carcinoma of the esophagus. In the early stages it is mild and intermittent. Later, it becomes continuous and progressively more severe. Pain is a late symptom and indicates extension of the growth into the surrounding tissues. Hemorrhage may occur but is seldom massive. Because of regional extension involving one or other of the main respiratory passages, pulmonary symptoms may overshadow those caused by the esophageal lesion. Voice changes, due to involvement of the recurrent nerves, frequently point to inoperability.

The differential diagnosis must exclude esophagitis, benign stricture of the esophagus,



CHAPTER XVIII

THE STOMACH, DUODENUM AND SMALL INTESTINE

DONALD R. WEBSTER, M.D.

STOMACH

Introduction

Primitive man was probably more aware of his stomach than any other internal organ. All the disturbances in his peritoneal cavity, as well as hunger and satiety, were interpreted by the reaction of the stomach. Despite this, our knowledge has grown very slowly and it has been difficult to correlate physiological, clinical, and pathological information. As all good surgery must rest on a sound anatomical and physiological basis, so gastric surgery has progressed only with advances in such fundamental knowledge.

It was the United States army surgeon, William Beaumont, in 1825, who had the imagination and perseverance to study Alexis St. Martin, the Canadian *voyageur* who developed a gastric fistula from a gunshot wound. This is one of the most fascinating stories in medical experience and laid the foundation of gastric physiology. Beaumont collected gastric juice, described the properties, and recognized the influence of emotions on the circulation, secretion, and motility of the stomach. This fundamental work has its modern counterpart in a monograph by Wolff and Wolff describing their subject "Tom," and confirming and extending Beaumont's original observations. The great Russian physiologist, Pavlov, devised the innervated gastric pouch, demonstrated the psychic phase of gastric secretion and proved the vagal pathways of the stimulus. Many modern physiologists, such as Babkin, a pupil of Pavlov's; Cannon, Ivy, Carlson, and others, have gradually extended our knowledge, but much remains to be done. We do not know exactly how the hydrochloric acid of the gastric juice is formed, why the stomach

does not digest itself, how painful sensations in the stomach are transmitted, and why ulcers form and heal in some persons and not in others.

Embryology.—The stomach appears in the fourth week of embryonic life as a spindle-shaped dilatation of the primitive gut, caudal to the lung bud. Just distal to this the liver bud appears and from this two dilatations arise which form the gall bladder and the anterior part of the pancreas (the posterior portion of the pancreas arises as a separate bud). There are both dorsal and ventral mesenteries, the latter, however, extending only as far as the umbilicus. The dorsal border and esophageal end of the stomach grow more rapidly than the pyloric end, and a rotation occurs. The dorsal surface turns to the left, and the ventral and pyloric end to the right, following the growing liver. Thus the ventral side becomes the lesser curvature, the dorsal surface the greater curvature, the left side the anterior surface, and the right side the posterior surface. The vagus nerves follow the rotation so that the left becomes anterior and the right posterior. The dorsal mesentery forms part of the lesser sac and the great omentum. The ventral mesentery by the growth of the liver forms the lesser omentum and falciform ligament.

Anatomy

The stomach varies in size and shape, and must be viewed with the fluoroscope to visualize the individual differences. The cardia is fairly constantly situated at D12 and the pylorus at L1 or 2. The position of the greater curvature, however, depends on the state, shape and content of the stomach. The organ is described as steer-horn or J-shaped and any gradation may occur be-

benign tumors, cardiospasm and bronchogenic carcinoma

Diagnostic procedures include x-ray examination, esophagoscopy, to directly visualize the growth and obtain a biopsy, and bronchoscopy to exclude involvement of the left main bronchus when x-rays have shown the lesion to be in that vicinity. A skeletal x-ray survey must be done to rule out metastases to the long bones and pelvis.

Treatment.—The management of carcinoma of the esophagus depends upon the extent of the lesion. Because of involvement of surrounding structures, the growth is frequently not resectable. In this event, gastrostomy is performed as a purely palliative procedure. If removal of the growth is considered feasible, the objective is its wide resection together with regional lymphatic glands and re-establishment of the continuity of the alimentary tract by esophago-gastrostomy. In recent years, the thoracoabdominal approach has enabled successful anastomoses to be performed as high as the first cervical segment of the esophagus. Previously, multiple stage procedures were more commonly used. These comprised esophagostomy, gastrostomy, resection of the lesion and, finally, the joining of the upper esophageal segment to the stomach by means of extra-thoracic tubular skin grafts. The procedure most favored is that of esophago-gastrostomy. By this method the stomach is mobilized and brought into the left pleural space. Here it is anastomosed to the proximal end of the esophagus. A more recent procedure involves removal of the esophageal lesion and anastomosis of the jejunum to the proximal esophageal segment. The jejunum is brought to the proximal portion of the esophagus through a tunnel which is easily made in the loose tissue lying just beneath the sternum.

Results of surgery of the esophagus are related to two factors:

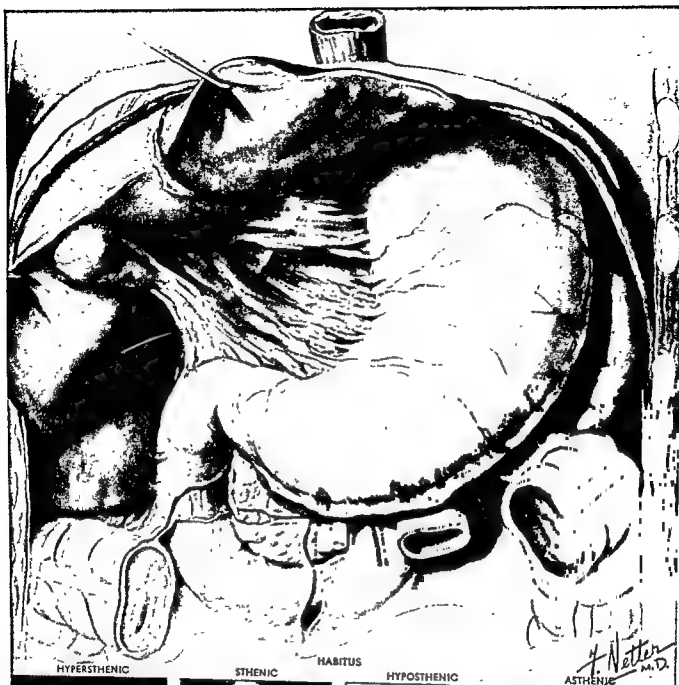
1. The extent of the disease process
2. The location of the lesion.

In general approximately 68% of the cases seen by the surgeon are suitable for surgery. Resection is possible in about 78% of these cases, that is, in 53% of all cases. If cases of carcinoma involving the cardiac end of the stomach are included, resection can be carried out in 70% of all cases encountered.

The immediate operative mortality rate is 14% for lesions below the aortic arch and 33% for those located above this level. The average operative mortality varies from 20 to 25%. Long-term survival statistics indicate that if one includes all types of carcinoma of the esophagus, there is an overall 24% three-year survival rate with a 10% five-year survival rate.

REFERENCES

- Adams, W. E., and Phemister, D. B.: Carcinoma of Lower Thoracic Esophagus, Report of Successful Resection and Esophago-gastrostomy, *J Thoracic Surg* 7: 621-632, 1938.
- Churchill, E. D., and Sweet, R. H.: Transthoracic Resection of Tumors of Stomach and Esophagus, *Ann Surg* 115: 897-920, 1942.
- Garlock, J. H.: Surgical Treatment of Carcinoma of Thoracic Esophagus, *Surg, Gynec. & Obst* 70: 556-569, 1940.
- Haight, C.: Congenital Atresia of Esophagus With Tracheoesophageal Fistula, *Ann Surg* 120: 623-655, 1944.
- Harrington, S. W.: Pulmonary Diverticulum of Hypopharynx at Pharyngo-Esophageal Junction, *Surgical Treatment in 140 Cases*, *Surgery* 18: 66-81, 1945.
- Kinsella, T. J., Morse, H. W., and Hertzog, A. J.: Spontaneous Rupture of Esophagus, *J Thoracic Surg* 17: 613-631, 1948.
- Ladd, W. E.: Surgical Treatment of Esophageal Atresia and Tracheoesophageal Fistula, *New England J Med* 230: 625-637, 1944.
- Ladd, W. E., and Swenson, O.: Esophageal Atresia and Tracheo-esophageal Fistula, *Ann Surg* 125: 23-40, 1947.
- Lahey, Frank Howard: Pharyngo-esophageal Diverticulum, Its Management and Complications, *Ann Surg* 124: 617-636, 1946.
- Sweet, Richard H.: Advances in Surgery of the Esophagus, *Advances in Surgery* 2: 41-80, 1949.
- Sweet, R. H.: Carcinoma of Midthoracic Esophagus, *Ann Surg* 124: 653-666, 1946.
- Sweet, R. H.: Pulmonary Diverticulum of Pharyngo-esophageal Function, *Technic of One-stage Operation*, *Ann Surg* 125: 41-48, 1947.
- Torek, F.: First Successful Case of Resection of Thoracic Portion of Esophagus for Carcinoma, *Surg, Gynec. & Obst* 16: 614-617, 1913.



HYPERTONIC STOMACH



ORTHOTONIC STOMACH



HYPOTONIC STOMACH



ATONIC STOMACH

tween these extremes depending on the habitus of the patient and the tone of the stomach at the time of examination

The stomach is divided into cardia, fundus, body and pylorus. The cardia is the area surrounding the esophageal opening. The fundus extends to the left and about one inch above the level of the cardia is in contact with the left dome of the diaphragm. The body extends roughly from the fundus to the incisura angularis. The pyloric antrum is that portion between the incisura and the duodenum

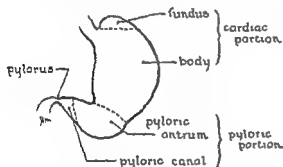


Fig 158.—Regional anatomy of the stomach

The wall of the stomach consists of a serosa of peritoneum, three muscular layers, and a mucosal lining. The serosa covers the stomach except at the cardia and at the lesser and greater curvatures where it separates to form the lesser and greater omenta. Of the three muscular coats, the external longitudinal layer is continuous with that of the esophagus above, and the pyloric sphincter below. Its fibers are scarce over the anterior and posterior surfaces. The middle circular layer covers the whole stomach and ends as the pyloric sphincter. The inner oblique layer is continuous with the circular muscle layer of the esophagus and is well developed over the fundus of the stomach and fans out toward the incisura

The mucosa covers the whole of the interior of the stomach. There is an abrupt change in its character at the beginning of the pylorus that can often be seen with the naked eye. Here the peptic glands are

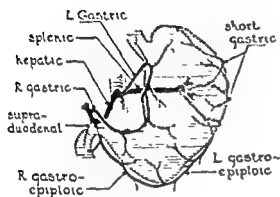
gradually replaced by the pyloric glands that secrete only an alkaline mucus. The peptic cells form compound racemose glands that secrete several substances. The goblet cells of the surface secrete the visible mucus. The neck cells probably secrete the dissolved mucin. The chief cells containing zymogen granules secrete the enzyme pepsin, and oxyntic cells hydrochloric acid. Scattered through the gland are the argentaffine cells whose function is not known but may be related to the formation of the intrinsic factor of Castle. Over all is a squamous epithelial layer continuous with the duodenum, the layer of Duran-Jura.

Arterial Supply.—The entire blood supply of the stomach comes from branches of the celiac axis. The left gastric artery runs toward the cardia in the lesser omentum, supplying branches to the esophagus and lesser curvature and ends by anastomosing with the right gastric artery. The right gastric artery, a branch of the hepatic artery (occasionally of the gastroduodenal artery), passes to the left along the lesser curvature to anastomose with the left gastric artery. The hepatic artery also gives off the gastroduodenal which runs below the pylorus and first part of the duodenum and divides into the superior pancreaticoduodenal and right gastroepiploic artery that runs to the left in the greater omentum giving branches to the greater curvature. The left gastroepiploic arises from the splenic artery, giving branches to the greater curvature and greater omentum. Occasionally this artery anastomoses directly with the right gastroepiploic artery, but more often there is a gap of several centimeters between the two vessels, and the anastomosis is through the smaller branches.

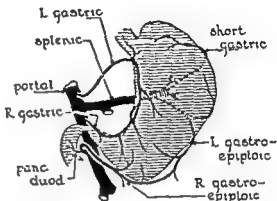
Veins.—The venous drainage is almost entirely into the portal system. The right gastroepiploic drains into the superior mesenteric vein, a tributary of the portal, while the gastric and splenic veins empty directly into the portal vein. The vicinity of the

cardia and lower esophagus is one of the most important communication areas between the systemic and portal systems. Esophageal varices may develop from any obstruction in the portal and splenic venous drainage.

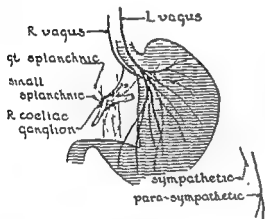
the plexus of Meissner and Auerbach. The right vagus nerve, now the posterior, sends branches to the celiac ganglion and breaks up to supply the posterior wall of the stomach, pylorus, and probably the whole intestinal tract, through the intrinsic plexuses



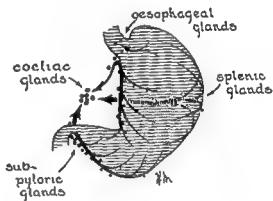
Arteries



Veins



Nerves



Lymphatics

Fig 159 — Arteries, veins, nerves, and lymphatics of stomach

Nerve Supply.—The stomach is supplied by branches from the parasympathetic system through the two vagi, and from the sympathetic system by branches from the celiac ganglion that accompany the blood vessels. The left vagus becomes the anterior vagus below the diaphragm, dividing into a number of branches. Some of these fibers anastomose with the right vagus, but the majority supply the anterior wall of the stomach and the lesser curvature. These disappear beneath the serosa and enter into

The celiac ganglion receives efferent fibers from the greater and lesser splanchnic nerves. Thus the stomach has a double nerve supply. The vagus is the main secretory nerve of the stomach, but in most other functions the two systems act in a reciprocal or supplementary manner.

Lymphatics.—The lymph gathers into radicals in the submucosa. These pierce the muscles and serosa, forming large vessels which in general follow the course of the veins. Thus the lymphatics of the lesser

hormone appears to be formed mainly in the mucosa of the pyloric portion of the stomach, and it is for this reason that the pylorus should be removed when doing a subtotal gastrectomy. If it is left, gastrin may continue to be formed and the resulting secretion from the remaining portion of the stomach will predispose to the formation of another ulcer. A similar hormone though less active (*enterogastrin*) is formed in the duodenum and upper jejunum

and called "enterogastrone." There are other gastric secretory depressants formed in the intestinal tract, and Gray has extracted an inhibitory substance from the urine of normal persons termed *urogastrone*. Whether these substances play any part in the normal regulation of gastric secretion is not known.

A diminished secretion and achlorhydria occur frequently in older persons, in diabetes mellitus, and in carcinoma of the stomach. Complete achlorhydria is always present in

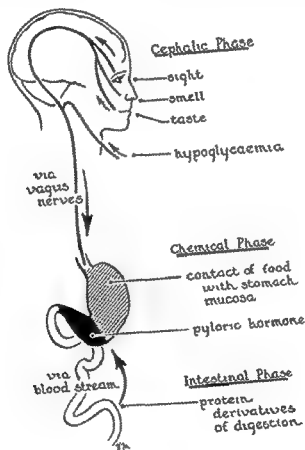


Fig 160 —Mechanism of the stimulation of gastric juice secretion.

Meat and protein extracts are the strongest stimulants of gastric secretion, while carbohydrates have very little effect. Fats stimulate a secretion of gastric juice while in the stomach but after passing into the duodenum stimulate the formation in the intestinal mucous membrane of a substance causing marked inhibition of gastric secretion. This substance has been extracted by Ivy

pernicious anemia. It is also found in some cases of hyperthyroidism, Addison's disease, arthritis and some of the dermatoses.

The stomach exhibits tone and peristalsis. The tone keeps the stomach wall closely applied to the food mass. The peristaltic waves gradually move the liquefied bolus to the pyloric antrum and into the duodenum. There have been many suggestions as to the

curvature drain away from the pylorus, following the coronary vein to the celiac glands and communicating in their course with the lower esophageal lymphatics. There are numerous glands scattered throughout the lesser omentum. The lymphatics on the greater curvature are much fewer in number than on the lesser curvature. The fundus and upper part of the body drain to the hilus of the spleen, while the lower portion and pylorus empty into the subpyloric group and into the celiac glands. The lymphatics of the pylorus and duodenum do not communicate, thus hindering the distal spread of malignant disease from the stomach.

Physiology

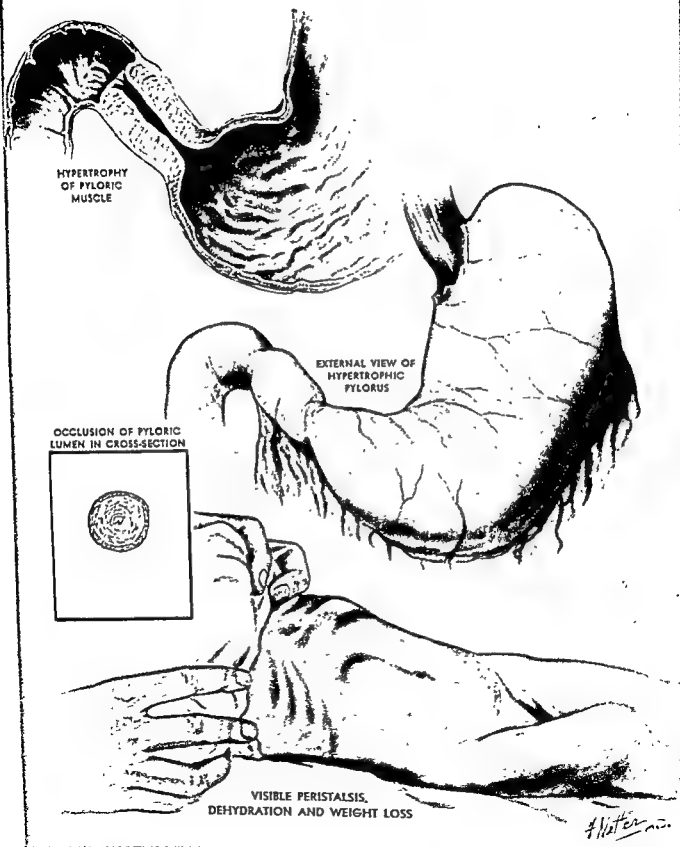
The stomach in addition to being a reservoir has important secretory and motor functions. The stomach secretes hydrochloric acid which was first identified by Prout in 1824. Despite intense investigation the exact mechanism of its formation is not known but is probably the result of a complicated enzymatic process. The hydrochloric acid concentration of pure gastric juice is about pH 1. It is probable that the parietal cells secrete the acid at the same concentration, and variations in acidity are due to diluting and buffering agents such as saliva, food, and mucus. The function of the acid is to provide an optimum pH for the action of the proteolytic enzyme pepsin. Hyperacidity is due to hypersecretion with less opportunity for dilution. Pepsin is secreted by the so-called chief cells. These lie scattered throughout the body of the gastric gland and in the resting state are full of granules that disappear with secretory activity. Pepsin is probably secreted in an inactive state as propepsin that passes into the lumen of the gland to be activated by the hydrochloric acid. The propepsin is also absorbed into the circulation and excreted in the urine as uropepsin. This can be measured and is reported to be increased in peptic ulcer cases, especially accompanying exacerbations of ulcer activity.

Mucus is secreted in two forms, a visible material which is a product of the surface epithelium and a dissolved mucus secreted by the neck cells. This latter substance contains a mucoprotein probably associated with the so-called intrinsic factor that combines with Vitamin B₁₂ to produce a substance essential for hematopoiesis.

The secretion of gastric juice consists of three phases: cephalic, chemical, and intestinal. The cephalic or *psychic phase* is initiated by the sight, smell, taste or thought of food. The stimulus passes by the vagus nerves to the gastric glands, and this is abolished by atropine and vagotomy. The secretion produced by vagal stimulation accounts for about one-half of the total secretion of the digestive period. It is much richer in enzymes than the secretion resulting from the chemical phase.

Hypoglycemia stimulates the vagal centers, producing a large flow of gastric juice. This is used as a test for completeness of the operation of vagotomy. Insulin is given in sufficient amounts to reduce the blood sugar to at least 50 mg. %. If no secretion occurs in the stomach, it can be assumed that all vagal fibers have been interrupted, providing a control test produced a secretion before the operation. The lesser curvature of the stomach has a more active vagal innervation than the greater curvature. Stimulation of the sympathetic fibers has a slightly inhibitory effect on the secretion produced by vagal or chemical means. There is evidence that the vagal stimulus exerts its full action only if the pylorus is intact and conversely chemical stimulation of the stomach is reduced if the vagi are cut. This suggests that there is an interdependence in their activities.

The two succeeding phases are the physiological sequelae to the psychic secretions. The presence of food in the stomach results in the formation of a hormone (*gastrin*) that produces a flow of juice, of high acidity but poor in enzymes. This was considered at one time to be histamine, but Komarov proved it to be a separate entity. This hor-



emptying mechanism of the pyloric valves, such as the acidity of the duodenal contents or the relaxation before an advancing peristaltic wave. Recent work suggests that the pylorus is normally in a patulous state and when the stomach contents are sufficiently liquefied and diluted they pass into the duodenum. The bolus remains for a few moments in the first part of the duodenum, to allow perhaps for neutralization. This area is known as the *duodenal cap* when visualized by x-ray, failure to outline this region or demonstration of a deformity is often due to the presence of an ulcer.

The vagus is usually considered as motor to the stomach and the sympathetic as inhibitory, but either nerve may produce the same motor effect depending on the state of the organ when the stimulus arrives. No "pacemaker" that initiates or controls peristaltic movements has ever been demonstrated.

CONGENITAL ANOMALIES

Congenital anomalies of the stomach are rare and, except for congenital hypertrophic pyloric stenosis, may be considered as anatomical curiosities.

Anomalies of Position

Absence and duplication of the stomach have been described. A portion or all of the stomach may be in the thorax as the result of a defect in the diaphragm. This may also be the result of a congenitally short esophagus or a hiatus hernia. Occasionally an ulcer develops at the point where the stomach is adherent to the margins of the defect. Failures of rotation and transposition of viscera are uncommon.

Anomalies of Structure

Atresia of the cardia and the pylorus are extremely rare. Diverticula occur occasionally and contain all coats of the stomach; congenital hourglass deformities have been

reported. Hourglass deformities are usually the result of scar contraction about a saddle ulcer.

Congenital Hypertrophic Pyloric Stenosis

This condition, as the name implies, is a hypertrophy of the circular musculature of the pylorus in infants, causing a stenosis of the pyloric canal. It has been recognized as an entity since 1773 when Armstrong reported three cases, and since then has been extensively studied.

Etiology.—It is said to occur in 0.5 to 1.0% of all infants, although not severely enough in all cases to require treatment. It is commoner in the male (7:1), the first born, and may affect several members of the same family. It is occasionally seen in adults and may be the persistence of this lesion. No etiological agent has ever been discovered, although an autonomic imbalance similar to cardiospasm is a probability.

Pathology.—The disease affects the circular muscular layer of the pylorus, which may be enlarged to many times its normal size. The tumor produced is usually palpable through the abdominal wall. There is edema of the mucosa and spasm associated with the hypertrophy, which contribute to the obliteration of the canal. The tumor is never fixed and the serosa is intact. There may be hypertrophy and dilatation of the stomach as the result of the obstruction.

Symptoms and Signs.—The typical clinical picture is the onset of regurgitation, then projectile vomiting in a previously normal infant. This usually occurs in the second to fifth week of life. Feedings are often well retained for a short time, but soon vomiting occurs after meals and the signs of starvation and dehydration appear. The child loses weight, the eyes become sunken, and the skin wrinkled and loose on the body. Bowel movements are small and the urine scanty. Occasionally periods of remission may occur and feedings are retained for a time. This is apparently due to a temporary relaxation of the pyloric muscle. The

vomit does not contain bile. Gastric peristaltic waves, sometimes very pronounced, are usually observed. The presence of a tumor in the right upper quadrant, palpable

when the child is being fed. The enlarged pylorus is felt as a smooth, firm, oval mass, that can be manipulated by the hand from beneath the liver or rectus muscle. There

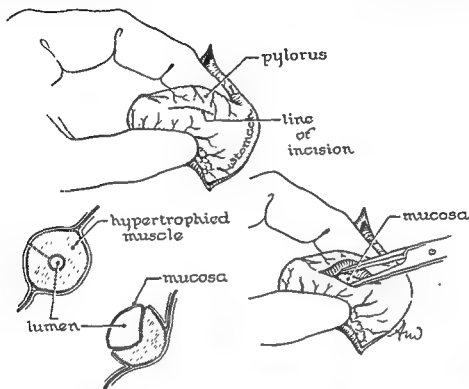


Fig 161 —Fredet-Ramstedt operation for hypertrophic pyloric stenosis

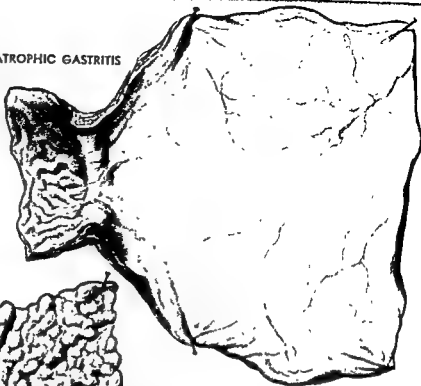
in over 90% of cases when the child is relaxed, and sometimes visible, confirms the diagnosis. This is more easily demonstrated

is no nutritional edema despite starvation. Alkalosis may occur because of the prolonged loss of hydrochloric acid.

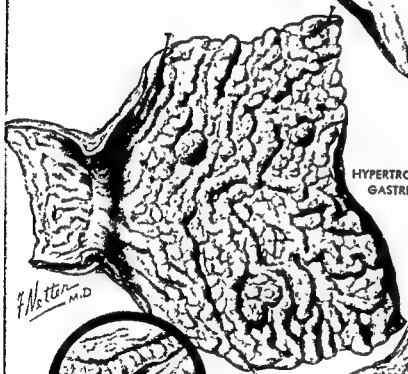


GASTROSCOPIC VIEW

ATROPHIC GASTRITIS

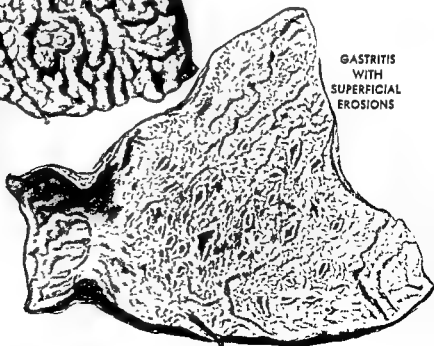


HYPERTROPHIC GASTRITIS



F. Netter M.D.

GASTRITIS
WITH
SUPERFICIAL
EROSIONS



the stomach, forming a perfect cast. The symptoms are a sense of fullness, foul breath; pyloric obstruction may ensue.

Occasionally penetration of the stomach wall may occur from foreign bodies in the abdominal cavity or neighboring organ such as instruments, drains, or gallstones.

Treatment.—If the articles are small they will be passed. It is remarkable how large an object will go through the pylorus and ileocecal valve. Such things as open safety pins, needles, razor blades, should be observed with the x-ray, and if not passing satisfactorily should be removed surgically.

INFECTIONS OF THE STOMACH

Acute Infectious Gastritis

An inflammatory reaction of the gastric mucosa accompanies many febrile conditions. It is marked by burning epigastric pain, nausea, and vomiting. Achlorhydria may be present. Occasionally hemorrhage occurs which may be confused with bleeding ulcer. The condition disappears as the systemic condition improves.

Acute Suppurative or Phlegmonous Gastritis

Phlegmonous gastritis is a very rare condition in which a part or the whole of the stomach wall may be involved in an acute suppurative process.

It occurs most frequently in the course of some septic process elsewhere in the body but may follow ulceration or trauma of the stomach. The streptococcus is the commonest organism involved, but staphylococci, pneumococci, and the anaerobic groups have been found.

The process spreads in the submucosa, pus collects, and the whole mucous membrane of the stomach may be shed as a cast. The symptoms are those of an abdominal catastrophe with agonizing, steady, epigastric pain, nausea, vomiting, rigidity, high fever, and collapse. Recovery may take place, but death usually ensues due to intoxication, or

perforation with peritonitis. The differential diagnosis includes perforation, acute pancreatitis, and cholecystitis. Treatment consists of antibiotics, intravenous alimentation, and drainage of any localized abscess. Healing may be followed by a form of *linitis plastica*.

Acute Gastritis

Acute gastritis may result from the ingestion of corrosives or highly irritating substances. The extent of the reaction depends upon the corrosive action of the material and varies from congestion and edema to ulceration, necrosis, sloughing and perforation. If the poison has not resulted in a marked corrosive effect on the pharynx and esophagus, or if its character is known, the stomach should be emptied by lavage or emetics. The appropriate antidote should be administered, followed by catharsis. The patient may need circulatory support and sedation.

A milder form, sometimes referred to as exogenous gastritis, may occur with the taking of alcohol, extremely hot or cold beverages, bacterial products and some medicines such as quinine, iodine, bromides, etc. The symptoms are epigastric discomfort, burning, nausea, anorexia, vomiting and diarrhea. Achlorhydria is common in the acute stage and acidity gradually returns as the condition improves.

Treatment.—Usually vomiting has emptied the stomach. Sedation with an opiate is often required, followed by an antacid mixture. Return to a normal diet should be gradual.

Chronic Gastritis

Since the development of the flexible gastroscope by Schindler and others, gastritis has been diagnosed more frequently. Chronic gastritis may be loosely classified as hypertrophic, atrophic and mixed types. In the hypertrophic type, the rugae are large and coarse and often the site of erosions that bleed easily. There is proliferation of the

Diagnosis is made on the basis of an accurate history, visible gastric peristalsis passing from left to right, and a palpable tumor. Occasionally a barium meal is necessary to confirm the diagnosis. The only other conditions that simulate it are congenital bands and atresia of the duodenum. In atresia the vomiting usually occurs as soon as feedings are begun and may contain bile if the obstruction is below the opening of the common duct.

Treatment.—Once the diagnosis has been made with reasonable certainty, the child should be prepared for operation. Dehydration and starvation are relieved by parenteral fluids (15 cc of 5% glucose solution per pound of body weight, 10 cc. of normal saline per pound of body weight). Small blood transfusions of 10 cc per pound are helpful.

As soon as the child's condition permits, the operation is carried out. Today this is invariably a Fredet-Ramstedt procedure. A right paramedian or subcostal incision about 2" in length is made, the liver is retracted, and the hypertrophied pylorus is picked up. The peritoneum and a few muscle fibers are cut. The remaining fibers are then separated by a pair of small forceps or the handle of the scalpel. When the mucosa is exposed, the muscle is well separated by spreading artery forceps, taking care not to injure the mucous membrane which is left intact. If the mucosa is accidentally torn, it must be repaired immediately. Infants tolerate this procedure excellently if properly handled and the cure is permanent. Feedings may be begun as soon as the child is fully conscious.

TRAUMA, WOUNDS, AND FOREIGN BODIES INVOLVING THE STOMACH

Contusion

The stomach may be contused in association with other organs in crushing injuries. There is usually epigastric pain, blood-

tinged vomitus, and rigidity of the abdominal wall. It may be difficult to be sure there is not a perforation, and the patient must be watched carefully. The abdomen should be x-rayed to exclude the presence of free gas in the peritoneal cavity suggesting a perforated viscus.

Rupture

Rupture usually results from a blow to the abdomen when the stomach is full of food or distended with gas. An old ulcer may rupture or a small split occur near the cardia. The signs are those of a perforated viscus and the patient should be operated on as soon as conditions permit.

Wounds

Gunshot and knives are the commonest agents producing wounds of the stomach. The direction of the missile can usually be estimated and often there is injury to other viscera. If there is no sign of exit of the missile, its position should be localized with x-ray. Operation should then be carried out, not to remove the bullet, but to repair the viscus, and the surgeon must be prepared to deal with any injuries that may be found in other organs.

Foreign Bodies

Innumerable varieties of foreign bodies have been found in the stomach. They can be generally classified into those swallowed.

1 **Accidentally.** These are usually small and will be passed without trouble, such as needles, pins, buttons, coins, teeth, marbles, etc.

2 **Intentionally.** This usually happens with psychotic patients, professional "sword swallows," attempted suicides, or those hoping to conceal evidence. Articles removed have included nails, glass, razor blades, toys, money, parchment, hair, etc.

3 **Accumulation of swallowed hair** is called a bezoar. Vegetable fibers may become entangled with the hair until it fills

From 1900 onward there appears to have been a marked increase in the incidence of peptic ulcers all over the world. Patterson states that 12% of the American population have peptic ulcers at some period of their lives. The site of the ulcer has shifted from the stomach to the duodenum so that duodenal ulcers are about 10 times as common as gastric ulcers and far more common in men than in women.

the midbrain could produce ulceration in the stomach and duodenum and many psychiatrists think that a similar process is involved in some psychosomatic states. This is the basis for the operation of vagotomy.

Another school, originally led by the distinguished pathologist, Virchow, believes that ulceration is a local disease due to some devitalization of the mucosa from trauma, gastritis, foci of infection, allergy, emboli, or

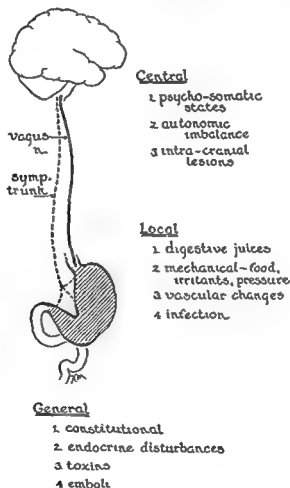


Fig. 162—Suggested etiological factors in the formation of peptic ulcers.

Etiology.—Peptic ulcer appears to be the result of conflict between constitution and environment. The proponents of the neurogenic concept believe that sustained emotional states cause increased secretion of gastric juice and alterations in the gastrointestinal mucosa permitting digestion of the tissue. Cushing demonstrated that lesions of

vascular change in the wall itself. Numerous attempts have been made to produce an experimental ulcer by irritation and trauma, but these lesions are usually acute and if they do not perforate, heal readily. One must guard against concluding from this that lesions of the gastric or duodenal mucosa which can be produced in animals by many

interstitial cells and lymph follicles while the submucosa is fibrosed. Mucus is often secreted in large quantities. In the atrophic type, the mucosa is thin and the glandular elements inactive. The muscle is atrophied and fibrotic. All gradations of the condition may occur, representing different stages of the disease.

It has not been our experience that any widespread gastritis is present in stomachs resected for duodenal ulcer, but is frequently present in cases of gastric ulcer. It is common in carcinoma of the stomach, which may account in part for the anorexia, achlorhydria, and epigastric discomfort.

Etiology.—There may be a definite and sometimes prolonged history of ingestion of irritants, notably alcohol, or hot, highly spiced foods, some drugs or infected material from the nose, throat or sinuses. It may also follow pyloric obstruction with stasis, or anastomotic operations. There may be a neurological mechanism involved. The atrophic variety is associated with some deficiency states, especially pernicious anemia when achlorhydria is invariably present.

Symptoms may vary from vague discomfort to an ulcer-like syndrome. The erosions may bleed, varying from a slight ooze to massive hemorrhage. There may be vomiting and in cases due to alcohol, the vomitus may contain large quantities of mucus.

Diagnosis is usually made by a careful clinical history and excluding other lesions by such examination as x-ray, gastroscopy and gastric analysis.

Treatment is directed toward removing underlying causes, and giving a bland diet. Hydrochloric acid should be given in the achlorhydria cases and antacid mixtures in the hypersecretory ones.

Tuberculosis

Tuberculosis of the stomach is rare and is almost always associated with tuberculosis elsewhere in the body. It may result from swallowed sputum, infected milk, or hema-

togenous spread from a distant focus. Direct extension from another organ may occur. The lesion may ulcerate or form a tumor with caseation in the submucosa which may then obstruct the pylorus.

Treatment.—The diagnosis is usually made following a laparotomy. Resection is not advised and the patient should be treated by supportive measures. The prognosis is poor.

Syphilis

Syphilis of the stomach is very rare and only occurs in patients with active disease. There may be an ulcer but usually the granulomatous process involves the stomach wall to produce the "linitis plastica" type of stomach. Symptoms usually simulate other lesions of the stomach and are not characteristic. The gastric crises of tabes may be a feature. The treatment is that of the lues although at times a total gastrectomy may be indicated.

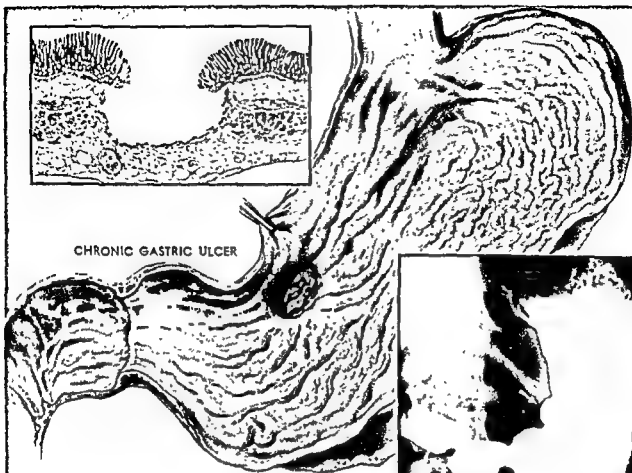
PEPTIC ULCER (GASTRIC AND DUODENAL ULCER)

Ulcerations of the stomach and duodenum have much in common both etiologically and pathologically, but appear to be different entities. They are often termed peptic ulcers and in many respects can be discussed together. They occur only in tissues in contact with acid gastric juice and therefore they are seen occasionally adjacent to aberrant gastric mucosa such as occurs in the lower portion of the esophagus, in a Meckel's diverticulum or in an anastomosis of the stomach to the jejunum.

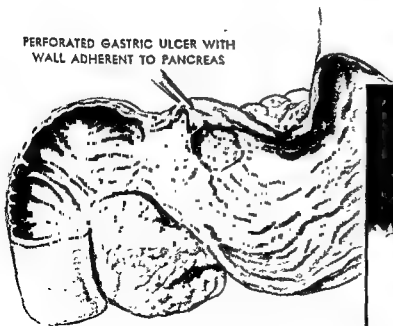
Peptic ulcer is apparently a disease of modern times. While the older writers occasionally mentioned the symptoms suggestive of peptic ulceration, no definite cases were described until the 18th century, and these were described as unique lesions. In the middle of the 19th century several small series of cases were recorded. These were mostly gastric ulcers. Duodenal ulceration was apparently regarded as a rare disease.



CHRONIC GASTRIC ULCER



PERFORATED GASTRIC ULCER WITH
WALL ADHERENT TO PANCREAS



F. J. Netter
M.D.

different types of insults have any relationship to the chronic ulcer in man.

Many lengthy series of ulcer cases have been analyzed and from these certain features are notable:

1. *Age* While peptic ulcers have been reported in the very young, duodenal ulceration seldom occurs before 20 years of age, and gastric ulceration still later. The age peak of duodenal ulcers is in the fourth decade, and gastric ulcers in the fifth.

2. *Sex* Peptic ulcers are about four times more common in men than in women.

3. *Occupation* In the Western world ulcers are more prone to occur in people with financial and administrative worries.

4. *Constitution and heredity.* Ulcer patients are usually the high-strung worrying type and appear to have a larger secretion of gastric juice than the normal person, especially the fasting and night secretions. There are numerous instances of peptic ulcers occurring in several members of the same family, which may indicate a predisposition to the disease.

5. *Diet.* There is no specific article of diet that can be proved to cause ulcer. There is some evidence that those who eat highly spiced foods, take alcohol on an empty stomach and those who cannot properly masticate food are more prone to develop an ulcer. Lack of a food factor such as vitamin B, as suggested by Sommerville, may play some part.

■ *Trauma.* Peptic ulcers occasionally occur in cases of severe trauma or burns and are called "Curling's Ulcers." These are probably due to the production of histamine-like substances from the traumatized area.

It is possible that not one but many etiological factors are responsible. There may be a deficiency of some protective substance represented perhaps by Ivy's enterogastrone or some basic endocrine or enzyme disturbance, such as an excess of lysozyme. Whatever the predisposing and initiating factor, it is the digestive action of the gastric juice

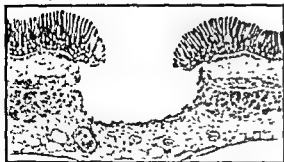
that actually produces the ulcers and prevents their healing. This has led to the dictum of "no acid, no ulcer," and the reduction of acidity below the optimum for the action of pepsin has been the aim of the most successful medical and surgical forms of treatment.

Pathology.—Gastric ulcers are usually situated in the pylorus or in the lesser curvature in the region of the incisura, occasionally high in the cardiac region. These are the areas where there are few, if any, acid secreting cells. Duodenal ulcers are most commonly seen in the first part of the duodenum, frequently so close to the pyloric ring that it is difficult to decide on which side the disease began.

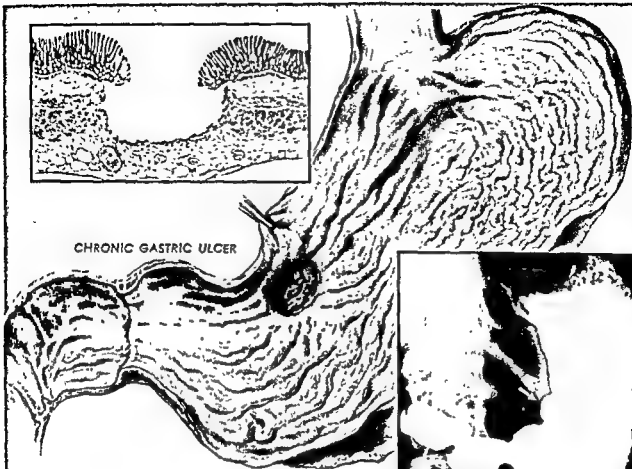
Peptic ulcers may vary from minute erosions to complete destruction of all coats of the wall. The ulcer itself may be shallow or deep, depending on its activity. The edges may be abrupt, shelving or undermining. The base soon becomes indurated and covered with fibrous tissue over which a gray slough can be seen. In large ulcers the base is often crossed by strings of mucous membrane. If situated posteriorly the ulcer may erode through so that the pancreas becomes involved and forms the base of the ulcer. If the ulcer is situated on the anterior wall, perforation may occur into the peritoneal cavity or it may become adherent to surrounding structures such as the liver or gall bladder. Bleeding may be minimal but if a large vessel is eroded, serious or fatal hemorrhage may occur.

In chronic ulcers there is always an inflammatory area surrounding the lesion, especially at the base. This may be marked with edema and induration. Vascular disturbances occur with thrombosis and obliteration of the arteries. Evidence of healing can often be observed by proliferation of the epithelium at the edge of the lesion and production of granulation tissues in the base.

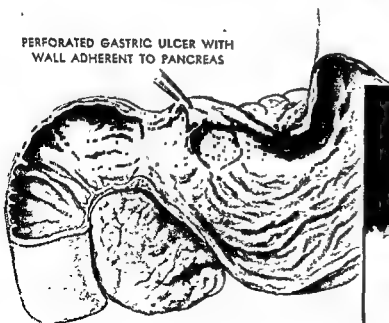
In the healing process cicatrization may cause serious complications. Those of the lesser curvature may give rise to "hourglass"

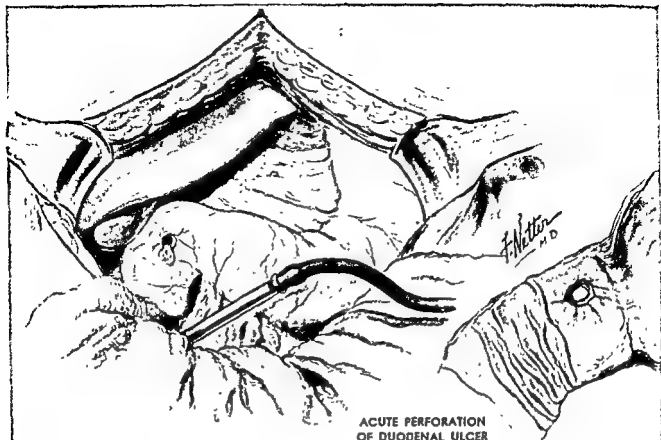


CHRONIC GASTRIC ULCER

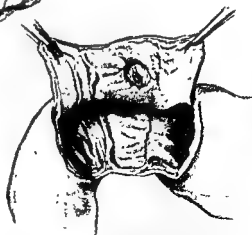
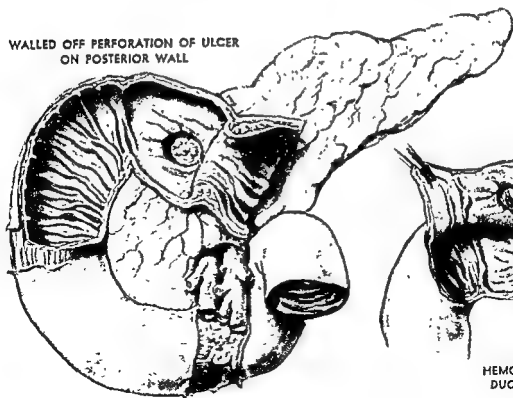


PERFORATED GASTRIC ULCER WITH
WALL ADHERENT TO PANCREAS





WALLED OFF PERFORATION OF ULCER
ON POSTERIOR WALL



PEPTIC ULCER

stomach and those near the pylorus to obstruction preventing the emptying of the stomach.

Clinical Picture.—Pain is the most distressing feature of peptic ulceration. It may vary from a feeling of slight discomfort to a gnawing, burning ache that intrudes into the daily life of the victim and disturbs his rest at night. It can produce a facies depicting suffering, and change a person into a hopeless hypochondriac. In the early stages of the disease the location of the pain is indefinite, but if long-continued and an inflammatory reaction is present, the gastric ulcer pain is in the midepigastrium and that of duodenal ulcer to the right of the epigastrium.

and also a poorly understood individual rhythm, where the patient may be completely free of pain for weeks or months. These are usually periods of transient healing when the crater may disappear in the x-ray picture. Attacks may be precipitated by worry, fatigue, alcoholic beverages and excessive smoking, and also by certain specific foods, especially coarse vegetables, fried food, which the patient soon learns to avoid. Realizing that food or alkalies will relieve the pain, he often carries some medication with him and to his bedside at night.

When the ulcer is active and penetrating anteriorly toward the serosa or posteriorly toward the pancreas, the pain is usually aggravated and assumes a boring, gnawing

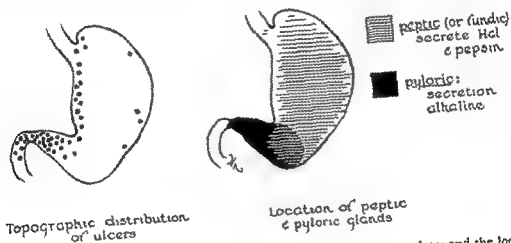


FIG. 163.—Diagrammatic comparison between the distribution of peptic ulcers and the location of peptic and pyloric glands.

The pain of duodenal ulcer appears some 2 to 3 hours after a meal, and is relieved by the taking of more food or alkalies. This sequence was described as the "ulcer rhythm" by Moynihan who epitomized it as gastric ulcer: food, comfort, pain, comfort, and duodenal ulcer: food, comfort, pain. The pain is seldom typical, however, and symptoms may be indefinite.

Sometimes the pain is accompanied by belching, nausea, and occasionally vomiting, which may relieve the pain. There is frequently a seasonal periodicity to the symptoms, increasing in the spring and autumn

character which may become continuous, unrelieved by food. This may suggest a dangerous change in the activity of the ulcer, often indicating a progression of the penetration. The mechanism of this pain is not satisfactorily explained. There are many factors that may influence the sensation of pain such as the size and location of the ulcer, the degree of penetration, the involvement of adjacent structures, and the pain threshold of the patient. However, the explanation of the pain in uncomplicated ulcer is not satisfactory. Such theories as the contact of hydrochloric acid with the ulcer sur-

face does not explain the presence of pain when the stomach is empty. Many investigators have attempted to correlate the hunger contraction of the stomach with the waves of pain in ulcer, but again the relation is not consistent. Others feel that *ulcers are only painful when an inflammatory action is present*. The instant relief produced by vagotomy may be explained on a diminished secretion of gastric juice or the reduction in the motor activity of the stomach.

Physical Signs.—There are no definite physical signs but ulcer patients are frequently though not always thin, having lost weight through a self-imposed diet. The face occasionally portrays suffering or irritability. Pallor may be present if bleeding or malnutrition has been prolonged.

Locally there is usually hyperesthesia over the epigastrium and tenderness on deep palpation. There may be some guarding of the muscles over the ulcer and tenderness over the lower dorsal vertebrae, particularly if the ulcer has involved the pancreas or posterior abdominal wall. If pyloric obstruction is present, a succussion splash can be elicited and visible peristalsis observed.

The palpation of a mass suggests a *marked inflammatory reaction about a penetration*, and malignancy must be excluded.

Diagnosis.—A careful history and observation of the patient are often adequate to suggest the diagnosis and confirmatory investigations can be carried out. Laboratory aids are most helpful, and with their proper utilization a high percentage of correct diagnoses can be obtained.

X-ray studies by means of a barium meal is the most useful diagnostic weapon we possess. This yields information about the *size and location of the ulcer, the patency of the pylorus, dilatation of the stomach, deformity of the duodenal cap or the presence of polyps or diverticula*.

Gastroscopy.—The gastroscope in the hands of experienced observers will fre-

quently demonstrate the site and the character of the lesion, if in the stomach, although the "blind areas" of the fundus limit its usefulness.

Gastric Analysis.—The examination of the stomach contents is at times a valuable aid in establishing a diagnosis. A tube is introduced into the stomach, and the fasting contents are withdrawn. If the volume is large, or if there are remains of food taken many hours previously, it is evidence of pyloric obstruction. A test meal of either food or alcohol which is seldom used now, or a chemical such as histamine or insulin, is given, and the contents are aspirated every fifteen minutes for two to three hours afterward.

The acidity and volume are measured; the presence or absence of blood is determined; and if there is a suspicion of malignancy, a cytological examination can be carried out. Large quantities of mucus suggest gastritis. If bile is present, the pylorus must be patent. The acidity and volume are usually higher in duodenal ulcer than in gastric ulcer cases, and very high levels suggest an active duodenal ulcer. The absence of free hydrochloric acid or a low acidity in the presence of a gastric ulcer is suggestive of malignancy.

Occult blood is usually positive in all active ulcers.

Treatment.—Surgery is required for those cases of ulcer that resist medical treatment, and where complications make it imperative. In uncomplicated ulcer, persistent pain despite adequate rest, diet, and medication is the symptom for which the patient demands relief. Every case, however, must be individually studied. The patient's background, his home, habits, and occupation must be reviewed to eliminate any adverse factors if a high percentage of success is to be attained.

The surgical procedure is planned to remove the lesion where possible, and to reduce the acidity to a point where peptic activity cannot take place. Gastric surgery has undergone a long and tedious develop-

ment, sometimes on a trial-and-error basis, following advances in our knowledge of gastric physiology. The earliest operations attempted closure of perforations. The next step was to direct the gastric contents into the jejunum because the pylorus was stenosed or to prevent the contact of chyme with the duodenal ulcer.

good, but a high percentage of recurrence followed after some years. Billroth, in 1881, performed the first partial gastrectomy, uniting the open ends of the duodenum and stomach. As this was not always feasible, he later anastomosed the jejunum to the posterior surface of the stomach. These are commonly spoken of as the Billroth I and

Royal Victoria Hospital Gastric Analysis

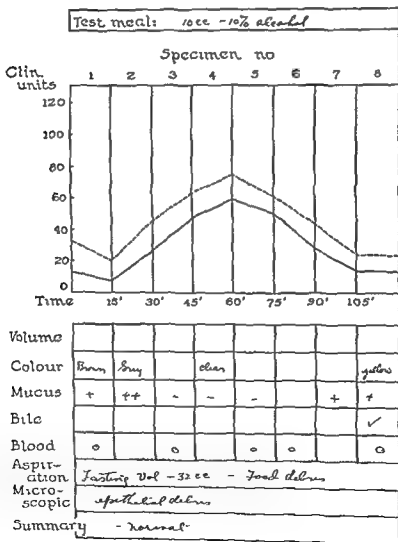


Fig 164—Sample chart of normal gastric analysis.

In 1881, anterior gastroenterostomy was described by Wolfier, and posterior gastroenterostomy by Von Hacker in 1885. The immediate results of the operation were

Billroth II operations. Later, in 1911, Polya demonstrated his method of anastomosing the jejunum directly to the open end of the resected stomach. This left too large a



perforation
with closure



anterior
gastro-enterostomy



posterior
gastro-enterostomy



wedge resection



sleeve resection



Billroth I



Billroth II



Polya (posterior)



Polya (anterior)



Hoffmeister-Finsterer



pyloroplasty



total gastrectomy

Fig. 165—Surgical procedures in the treatment of peptic ulcer.

stoma and various techniques for reducing the size of the opening and fashioning the anastomoses have been described by Balfour, Finsterer, Hofmeister and many others.

In cases of pyloric stenosis, pyloroplasties were performed by several surgeons whose methods still bear their names, such as Finney, Heineke and Mikulicz. These operations have now been abandoned in the treatment of peptic ulcer. Ligation of the majority of the gastric vessels was advocated by Wilkon Hey but has received little support in this country.

We believe the operation of choice is a subtotal gastrectomy removing about three-quarters of the stomach including the pylorus, and the ulcer site in the duodenum. The anastomosis is made posteriorly with a short loop of jejunum fixed high on the stomach so that angulation will not occur and with a stoma that will admit about two fingers. The pylorus should be removed because it is the main site of formation of gastrin, and, if left, acidity may persist and the ulcer recur. Occasionally, due to adhesions or activity, the ulcer cannot be removed and a procedure involving removal of the pyloric mucosa is carried out. This is known as an exclusion operation.

Vagotomy, either thoracic or subdiaphragmatic, disconnects the stomach from cerebral stimuli, thus abolishing the psychic phase of gastric secretion and reducing motility. Because of this latter effect and because there is often some degree of pyloric stenosis, it is advisable to do a gastroenterostomy at the same time to assist in evacuation of the stomach. Some surgeons are combining partial gastrectomy with a subdiaphragmatic vagotomy. Vagotomy is not recommended for gastric ulcer because of the possibility of malignancy. There is still some dispute as to the ultimate results of vagotomy. Some observers report a marked disturbance of gastric and intestinal motility that worsens with time. However, in other centers the results appear satisfactory if

combined with gastroenterostomy, and it is adopted as the procedure of choice in uncomplicated duodenal ulcer.

Complications.—The most serious complications of peptic ulcers are perforation, obstruction, and hemorrhage, and in the case of gastric ulcers, malignant degeneration.

Perforation

An ulcer that has penetrated through all coats of the organ is said to have perforated. This may occur slowly, and the inflammatory reaction about the lesion produces adhesions between it and the neighboring organs, so that general peritoneal soiling does not take place, and the adjacent tissue becomes the base of the ulcerative process. This is more likely to occur in posterior duodenal ulcers where the pancreas and posterior abdominal wall may be involved. In anterior ulcers, the liver, gall bladder or omentum may be the adherent organs. This process is sometimes referred to as subacute perforation. To differentiate such ulcers from the acutely perforated ones, the term penetrating ulcer is frequently used.

Occasionally a few drops of gastric contents may escape giving symptoms of acute perforation that rapidly improve, or if posteriorly, may form an abscess in the lesser sac. This is inaptly referred to as chronic perforation.

Acute Perforation

Acute perforation of an ulcer into the general abdominal cavity is one of the most dramatic of abdominal catastrophes. Frequently, without warning, sometimes following aggravation of ulcer symptoms, the patient is seized with agonizing epigastric pain that soon spreads over the abdomen. He lies on his back with his knees drawn up, the face pale, glistening with beads of perspiration and sometimes cyanotic. The patient often vomits. The temperature is subnormal, and the pulse is small but not usually over 100. The breathing is entirely

thoracic, and when one examines the abdomen, it is typically scaphoid, rigid or "boardlike," and tender. The tenderness may be more marked on the right side. X-ray or percussion usually reveals pneumoperitoneum, and the liver dullness is obliterated.

The initial shock usually passes off and the general condition and symptoms improve. The pain is eased, color improves, and the pulse is stronger. The abdomen, however, remains rigid and tender, and the respirations shallow. This is referred to as the stage of reaction which soon merges into that of general peritonitis with distention, hiccough, vomiting, and intestinal paralysis.

If the condition of the patient or circumstances do not permit immediate operation, the stomach should be kept empty by continuous suction, intravenous fluids should be given, and the pain controlled by sedatives. As soon as possible an attempt should be made to close the perforation. It is remarkable that about one-third of the patients who have had a perforation repaired by simple closure have no further ulcer symptoms.

Pyloric Obstruction

As most ulcers are near the pylorus on either the duodenal or stomach side, obstruction may result from spasm, edema, or cicatricial contraction in the healing process. As obstruction develops, the pain is not relieved by food or alkalies and vomiting becomes a prominent symptom, the vomitus often containing food eaten many hours before.

Dilatation of the stomach is a stimulus to secretion, and the resultant hypersecretion lost to the body because it cannot be absorbed results in alkalosis, hypochloremia and azotemia. X-ray with barium will demonstrate the dilatation and retention.

Continuous aspiration of the stomach contents and the administration of antispasmodic drugs will sometimes relieve the obstruction, indicating that it is due to spasm or edema. If obstruction persists, it is prob-

ably due to fibrotic contraction, and complete stenosis is an absolute indication for operation.

Hourglass Contraction

This deformity, dividing the stomach into two cavities, is due to a gastric ulcer, usually involving the lesser curvature. Previous perforations and perigastric adhesions or spasm may occasionally play some part, but the great majority are the result of reaction about a large, lesser curvature ulcer that involves both walls of the stomach—the so-called saddle ulcer. The fibrosis gradually produces a contraction that reduces the gastric pathway to a narrow channel close to the lesser curvature. The symptoms are usually those associated with the ulcer, although fullness, nausea, and regurgitation may be present.

Hemorrhage

Hemorrhage varies from a slight ooze, that can be detected as "occult blood" in the stools, to a profuse hemorrhage that can be fatal. Small amounts of blood lost continuously may produce a secondary anemia, although this is unusual in ulcer cases.

Hematemesis is the name given to the vomiting of blood. This may come from a peptic ulcer although other conditions such as esophageal varices must be considered. In duodenal ulcer the blood often does not enter the stomach but passes down the bowel to produce the black, so-called tarry stool, known as melena. If the passage is rapid, however, the blood may still be red in color. This is often the first indication that an ulcer is bleeding unless the hemorrhage is severe enough to produce weakness or fainting. Hemorrhage is loosely classified as leaking, gross, or massive, depending on its severity. The term massive hemorrhage is reserved for those cases that have sudden severe bleeding with sufficient loss of blood to produce signs of shock. Hemorrhage may be fatal in both the young and aged, but the older patients are more

prone to continued or recurrent bleeding because of sclerosis of the vessels and more liable to complications such as coronary and cerebral thrombosis.

Diagnosis.—Hemorrhage from an ulcer must be differentiated from bleeding from other lesions of the esophagus, stomach or bowel. The commonest are:

1. *Esophageal Varices.* This condition is due to some form of portal obstruction. It usually appears in middle age in association with such signs as enlarged liver and spleen and in some cases ascites

but they are usually easily recognized, and there has been no previous digestive history.

If the patient's condition permits, a small barium meal should be given, in order to exclude esophageal varices.

Treatment.—In the cases of occult and gross hemorrhage, a conservative course may be followed. The patient should be at rest with some sedation, and transfusions should be given to restore the blood loss.

Frequent feedings of a bland, high protein nature should be given with vitamin supplements. Cholinergic blocking drugs,

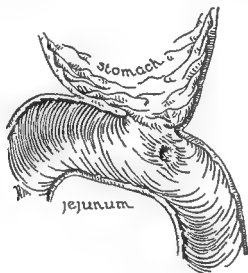


Fig 166 —Usual site of ulcer following gastrojejunal anastomosis

2 *Chronic Gastritis.* This condition sometimes causes massive hemorrhage, although frequently no gross lesion can be found. There are often numerous petechial, hemorrhagic areas, and the condition is sometimes referred to as gastrostaxis. There is usually a history of indigestion not typical of ulcer

3 *Cancer of the Stomach.* Simple hemorrhage usually occurs only late in the disease, although continuous loss of a small amount of blood may produce marked anemia.

4. *Blood Dyscrasias.* Such conditions as purpura, and leukemia, may cause bleeding from the gastrointestinal tract.

5. *Toxemias.* Scarlet fever, malaria, and some poisons may cause severe hemorrhage,

such as atropine or atropine-like substances appear to be of value.

The patient should be very carefully supervised with frequent recordings of blood pressure, pulse and hemoglobin. If bleeding recurs, especially in the older patient, operation should be advised.

In cases of massive hemorrhage with signs of shock, the patient should be at complete rest, with sedation and transfusions, preferably with fresh blood, begun as quickly as possible. The estimation of hemoglobin and red blood cells may not represent the true severity of the case because of hemoconcentration.

If the bleeding continues or recurs so that replacement is not keeping up with the loss,

a bold decision must be taken. This must be done before the patient's condition has deteriorated so far that operation is useless. Experience has shown that in patients who have had severe bleeding for 48 hours, any surgery is very hazardous. At operation, a direct approach must be made to the ulcer site, and the bleeding vessel ligated. Subtotal resection may then be carried out.

Stomal Ulcer

This distressing complication of gastrojejunostomy is fortunately rare if an adequate resection has been done. The incidence is probably about 1%. It follows operation for duodenal ulcer more frequently than for gastric ulcer. It is always situated on the jejunal side of the stoma. The lesion has the same pathological characteristics as peptic ulcer elsewhere and is subject to the same complications. It occasionally perforates into the general peritoneal cavity, but more often becomes surrounded by an inflammatory mass that becomes adherent to the colon and may form a fistulous connection, the so-called gastrocolic or gastrojejunocolic fistula.

Symptoms.—The symptoms of stomal ulcer are similar to those of ulcer of the stomach or duodenum but often more distressing. The pain is not related so characteristically to meals and is situated more in the midepigastrium or to the left of this area. Vomiting is frequent and may ease the discomfort. Bleeding may vary from a slight ooze to severe hemorrhage. Systemic signs of acute inflammation may be present and occasionally the inflammatory mass can be palpated.

Diagnosis is made on the history and demonstration of the ulcer by x-ray.

Treatment.—If the symptoms are not controlled by a strict medical regime, operation becomes necessary. The most popular method today is a vagotomy, because if successful it eliminates the more hazardous procedure of a secondary gastrectomy. This

latter operation may be very difficult if a primary posterior anastomosis has been carried out.

GASTROJEJUNOCOLIC FISTULA

Gastrojejunocolic fistula, as the name implies, is a fistulous connection between the stomach, jejunum, and transverse colon. Except in the rare case where a gastric ulcer perforates into an adherent colon, it is a complication of gastrojejunal ulceration. The colon and enterostomy site are usually bound together in an inflammatory mass, and the fistulous openings are quite close to one another.

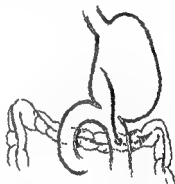


Fig. 167.—Gastrojejunocolic fistula.

Symptoms.—The symptoms are loss of weight, marked diarrhea, foul eructations, and occasionally true fecal vomiting. The diagnosis is confirmed by a barium enema and a barium meal. Both should be done as there may be a valvelike action in one direction that prevents proper demonstration of the lesion.

Treatment.—Medical treatment is of no avail and the patient will die of inanition unless surgical measures are taken. The patients, who are usually in poor condition, are brought into proper nutritional balance, and if conditions permit, a primary radical resection is carried out. If inanition is marked, a loop colectomy proximal to the lesion will lessen the diarrhea, and usually result in marked improvement in the patient's weight and general condition. The curative pro-

cedure is accomplished by freeing the colon from the mass and either closing the fistula or resecting the involved portion. The gastrotomy is then undone and the involved area of the small bowel is resected. This is followed by a higher gastrectomy and a new gastrotomy.

DUMPING SYNDROME

Under the heading of dumping syndrome is grouped a variety of symptoms that may occur after subtotal gastrectomy. These may include weakness, sweating, pain in the epigastrium, palpitation, nausea, vertigo, and fainting. Only some or all of these may be present. Most observers believe it to be due to distention of the jejunum and recommend the limitation of fluids and administration of atropine. Postprandial or reactive hypoglycemia has not been evident in many cases. There may be a functional element such as transference of symptoms after removal of the ulcer as the peripheral site of a psychosomatic lesion.

CARCINOMA OF THE STOMACH

Carcinoma of the stomach is responsible for one-fifth to one-quarter of all cancer deaths. It is rare in childhood, uncommon in early adult life, and almost twice as frequent in men as in women. Its insidious growth and trivial symptomatology in the early stages postpone the inevitable visit to the doctor, and even then the frequent vagueness of the complaints often results in palliative treatment for some time before the tragic presence of advanced carcinoma is demonstrated. Until every case of dyspepsia in patients over thirty-five years is suspect, and is examined radiologically, there will be little improvement in our results.

Etiology.—The cause of cancer of the stomach, like cancer elsewhere in the body, is quite unknown. Although heredity and familial predisposition to cancer are suggestive, a causal relationship is statistically difficult to prove. It is suggested that cer-

tain articles of diet may be involved, as cancer of the stomach is commoner in those countries where raw liquors are consumed and hot foods ingested, but factual proof is lacking. Experimental work has shown that cancer can be induced in animals by feeding fats and oils that have been superheated or "cracked" and thus become carcinogenic. It is very difficult, however, to produce an adenocarcinoma of the stomach in the experimental animal, and evidence is not sufficient to implicate any specific extrinsic agent.

There are, however, three conditions that should be considered precancerous or precursors of cancer of the stomach. These are gastric ulcer, gastritis, and some benign tumors, especially polyps.

Gastric Ulcers.—It has been conclusively demonstrated that cancer may develop in a benign chronic gastric ulcer. It is difficult to estimate the percentage but it is probably not high. Many ulcers thought to be benign are found to contain cancer cells when studied after removal. Finsterer reported a series where this was found to be about 20%. It is for this reason that any gastric ulcer that does not heal completely or recurs should be resected.

Gastritis.—While definite proof is lacking that gastritis is a precursor of cancer, the atrophic form of gastritis with achlorhydria, has a higher incidence of cancer than the normal or hypersecreting stomach.

Polyps.—Polyps of the stomach usually occur in association with achlorhydria and cancer-in-situ has been frequently found in them. While the malignant degeneration is not so apparent as in rectal polyps, they must be regarded as precancerous in a certain number of cases.

Pathology.—Two-thirds of gastric cancers arise in the prepyloric area. The lesion generally stops short of the pylorus proper and invasion of the duodenum is rare. Cancer of the lesser curvature is next in frequency and may arise high in the region of the

cardia. Only 5% of gastric carcinomas occur on the greater curvature, but ulcers situated in this area are almost always malignant.

Gastric carcinoma can be divided into four main groups.

1 The ulcerative form is the commonest and arises in the pyloric area or lesser curvature. It varies considerably in size and depth and characteristically has a hard everted edge with a dark red necrotic base which bleeds easily. Microscopically the groups of epithelial cells lie imbedded in abundant fibrous tissue. Infiltration tends to be rapid although the muscle coat is seldom completely destroyed. The neighboring lymph nodes are invaded very early. It may begin in an apparently normal stomach or at the edge of a chronic peptic ulcer.

2 The papillary or polypoid form is less common and presents as a sharply limited soft mass, projecting into the lumen of the stomach near the pylorus. It may represent malignant degeneration of a benign polypoid lesion. Enlargement is rapid and ulceration of the mucosa occurs early. As the tumor outgrows its blood supply, necrosis and infection may follow. Microscopically it is an adenocarcinoma but may assume the so-called encephaloid form with scanty stroma and spheroidal cells. It spreads slowly and may remain localized to the mucosa for a long time.

3. In the infiltrating type a fibrous thickening chiefly in the submucous and subserous coats may cause a stenosis of the pylorus. As the process extends, the body and finally the whole stomach may be involved. It is to this latter type that the term linitis plastica or leather-bottle stomach has been given. Ulceration may be present but is superficial. Microscopically it is a scirrhous cancer. Metastases are infrequent and occur late and this form offers a better prognosis than the other neoplastic lesions of the stomach.

4. Colloid or mucoid carcinoma is an adenocarcinoma but contains large quantities of mucus. It usually appears in the

pyloric region giving a gelatinous appearance to the thickened wall. It soon infiltrates the wall, producing colloid cancer of the peritoneum and the so-called "Krukenberg tumor" described below.

Spread.—Cancer of the stomach spreads by direct extension in the stomach wall, and to neighboring organs, by the lymphatics and by the blood stream. Since most carcinomas occur along the lesser curvature, the lymphatic drainage is to the coronary, hepatic and pyloric groups of glands. As the disease progresses, more lymphatics are invaded, creeping along the vessels to the celiac axis to eventually involve the aortic group and the glands in the hilus of the liver. Occasionally spread from the celiac glands may occur along the thoracic duct or by way of other mediastinal lymphatics to give rise to metastases in the supraclavicular area (Virchow gland). Invasion of the blood stream by malignant emboli may set up metastatic foci in the liver, lungs, bone, and brain in that order of frequency, but this is usually late. Lymphatic involvement of the liver is usually of a spreading pattern from the hilus, while blood borne metastases are scattered and often numerous.

Extension to the pancreas, liver, colon and omentum may occur when the growth finally penetrates the wall of the stomach. This is a late sequel, as the serosa appears to offer considerable resistance to the direct spread. Serosal involvement also sets cells free in the peritoneal cavity which may find a foothold on any peritoneal surface. The ovary appears to be particularly susceptible to transperitoneal implantation, producing a bulky secondary ovarian lesion (Krukenberg tumor) characterized by a mucoid cellular degeneration with displacement of the nucleus to form the "signet ring" appearance. If the cells lodge on the pelvic peritoneum, the so-called *rectal shelf* is formed, which can be felt on rectal examination.

Symptoms.—Unless the growth bleeds, or being near the cardia or pylorus causes

obstructive symptoms, cancer of the stomach is remarkably free of symptoms. It is for this reason that the onset of dyspepsia, commencing in a patient over forty who has been previously well, must be regarded as cancer until this has been disproved. Early symptoms are commonly a diminution in appetite and a feeling of distention after a meal. Recurrent attacks of nausea may occur. Pain is late and may be similar to that of peptic ulcer. Insidious hemorrhage usually gives rise to lethargy and pallor although massive bleeding may occur with a hematemesis or melena. Vomiting is uncommon in the early case unless there is obstruction to the pylorus, while with lesions in the cardia dysphagia is a prominent symptom. Only too often, however, these early symptoms are overlooked until marked cachexia and a palpable epigastric mass place the cancer in the incurable category. Any patient therefore who has dyspepsia, loss of weight or anemia occurring at or beyond middle age must have a complete examination of his stomach to exclude carcinoma, if he is to be saved by adequate surgery.

If there is a malignant degeneration in a previously benign gastric ulcer, there is a rather sudden change in the dyspepsia. The periodicity of the pain is lost, it becomes more persistent, and is not relieved by food and alkali.

Special Diagnostic Measures.—Gastric Analysis.—In approximately half the cases of cancer there is an achlorhydria or hypochlorhydria. However, the remaining cases show free hydrochloric acid within the normal range. Cytological examination of the aspirated material may reveal the presence of malignant cells.

Gastroscopic Examination.—In experienced hands the use of the gastroscope, which allows visualization of the tumor, makes the diagnosis certain in a percentage of cases and may also serve to exclude the diagnosis of cancer. However, the gastroscope has three blind spots: the fundus, the pylorus and the greater curvature opposite the cardia, which greatly limits its usefulness.

A negative gastroscopic examination, therefore, does not exclude the presence of carcinoma.

Occult Blood.—This is often positive and the test should never be omitted in any gastrointestinal investigation.

Roentgenography.—Careful radiological examination of the stomach with particular attention to the mucosal pattern will demonstrate the carcinoma in a high percentage of cases. However, repeated examinations may be necessary as the tumor may not be visualized in early cases.

Treatment.—The treatment of cancer of the stomach is entirely surgical. While past results have not been too encouraging, the survival rate is gradually rising due to improved technique and the widening of the criteria of operability. However, no large gain will be made until cases are diagnosed and treated earlier.

In cases involving the pylorus or body of the stomach, a wide subtotal gastrectomy should be done. The whole omentum and as far as possible the lymphatic drainage area must be excised as well. Occasionally portions of adherent organs such as pancreas, spleen or colon must be removed with the stomach. When the lesion lies near the cardia, a total gastrectomy through a thoracoabdominal approach is necessary.

Palliative resection, even in the presence of hepatic metastases, will frequently improve the comfort of the patient and prolong the survival period. Wangenstein and others have been practicing re-exploring patients after some months and removing any accessible metastatic glands. It is apparent that only early diagnosis and aggressive surgery will improve the prognosis of the cancer victim.

BENIGN TUMORS OF THE STOMACH

Such growths are uncommon and consist of adenomatous polyps which may be single or multiple. Submucous lipoma, fibroma, neuro- or myofibromas may occur. Pan-

creatic rests are occasionally found in the gastric wall near the pylorus

Adenomatous polyps may undergo malignant changes usually beginning at the periphery of the polyp. Tumors arising from the nerve sheaths are the neurolemmas and the neurofibromas, the latter sometimes being associated with generalized neurofibromatosis. Giant hypertrophy of the epithelium may occur giving the appearance of polyposis. Hypertrophy of the pyloric mucosa may act as a valve producing intermittent pyloric obstruction. The symptoms are usually vague epigastric discomfort, signs of obstruction or hemorrhage. Achlorhydria may occur. The treatment is surgical removal and total gastrectomy may be necessary in cases of diffuse polyposis.

DUODENUM

The name duodenum is derived from the approximate length of this portion of the bowel, the breadth of twelve fingers. It extends from the pylorus to the ligament of Treitz making almost a complete loop. It is arbitrarily divided into four portions

The first or superior portion, called by the radiologists the bulb or cap, is related above to the neck of the gall bladder and the undersurface of the liver.

The second part descends from the 1st to 3rd lumbar vertebra forming a curve about the head of the pancreas. It receives the common bile duct and main pancreatic (Wirsung) duct in the ampulla of Vater, and the accessory pancreatic (Santorini) duct about 1 to 3 cm above this. Posteriorly it rests on the right kidney, renal vessels, ureter, and psoas muscle

The third portion crosses transversely to the 3rd lumbar vertebra where as the fourth part, it turns abruptly forward to join the jejunum. This junction is supported by the ligament of Treitz. Posteriorly it is related to the vertebra, aorta, and left renal vessels.

Blood Supply.—The superior pancreaticoduodenal from the gastroduodenal branch of the hepatic artery anastomoses with the inferior pancreaticoduodenal, a branch of

the superior mesenteric artery. These vessels run along the concavity of the duodenum giving off numerous branches to the anterior and posterior surfaces. A separate branch from the gastroduodenal or the hepatic artery supplies the duodenal cap.

Innervation.—The duodenum, like the rest of the gastrointestinal tract, is innervated with sympathetic and parasympathetic fibers through the splanchnic and vagi nerves. These pass to the bowel in the coats of the blood vessels and probably act in a reciprocal fashion. Distention of the duodenum produces nausea and pain referred to the epigastrium.

Motility.—The duodenum exhibits peristalsis and rhythmic segmentation. The gastric peristaltic waves usually stop at the pylorus, but Ivy has described them as passing over to the duodenum. Reversed peristaltic waves are commonly seen with the duodenal contents regurgitated into the stomach.

Secretion and Absorption.—The duodenal mucosa, like that of the small intestine, has the remarkable ability to secrete and absorb simultaneously. Substances such as water, alcohol, and glucose are rapidly absorbed in isolated loops, but it is questionable whether this plays much part under normal conditions. Besides the digestive enzymes, the duodenum secretes such hormones as secretin, a form of gastrin, and other substances affecting the gall bladder, the regulation of gastric secretion and hematopoiesis which have not been completely worked out.

Congenital Anomalies

1. **Stenosis and atresia** may occur in any part of the duodenum, and the symptoms of high obstruction appear shortly after birth. If the stenosis is below the ampulla of Vater, the vomitus will contain bile.

2. **Malrotation.**—These anomalies have been grouped under three headings

(a) **Duodenum dextra.** This is usually associated with failure of

(b) *Duodenum mobile*. This is a persistence of a duodenal mesentery to any or all parts of the duodenum.

(c) *Duodenum inversum*. Here the second portion curves upward to the left.

These conditions are found in the course of investigation for other conditions. It is doubtful that many cases cause symptoms, but when such occur, they result from partial, intermittent obstruction, with crampy upper abdominal pain and vomiting of bile-stained material.

3 Bands and Membranes.—These peritoneal folds extend from the gall bladder and undersurface of the liver to the proximal portion of the duodenum. Such a fold is sometimes called Harris' membrane. Occasionally the membrane passes to a point below the duodenum, compressing it, giving symptoms similar to stenosis.

4 Diverticula.—These are out-pouchings usually from the second or third portions of the duodenum. They may be true diverticula involving all coats of the bowel or the longitudinal and circular coats may be missing. There is no peritoneal covering unless they are situated anteriorly. If the neck is wide, no symptoms are caused, but if narrow and the diverticulum is in a dependent position, food collects in the pouch and diverticulitis may result, giving epigastric distress and tenderness. If symptoms are severe, the diverticulum may be excised.

Duodenitis

Duodenitis may occur as an isolated entity or in association with gastritis. It may be part of an inflammatory or neoplastic process of neighboring organs such as the gall bladder or pancreas. It may be the result of stasis and dilatation caused by intermittent obstruction from congenital bands or malposition. It is seldom diagnosed.

New Growths

New growths of the duodenum are extremely rare. Excluding those of the am-

pulla of Vater, which may arise in the common duct or the rare case of extension of a pyloric or pancreatic cancer, duodenal tumors have an incidence of a fraction of 1%. The benign tumors include adenomas (polyps), myomas, lipomas, hemangiomas and pancreatic rests.

Malignant tumors are mainly carcinomas arising in the second part of the duodenum. Those near the ampulla of Vater may obstruct the flow of bile causing jaundice, which at first may be intermittent. It is usually confused with carcinoma of the head of the pancreas. The growth may eventually cause obstruction of the lumen of the bowel.

SMALL INTESTINE

Anatomy.—The small intestine is approximately twenty feet in length. It begins at the duodenojejunal junction and ends at the ileocecal valve. The upper two-fifths, the jejunum, occupies the left hypochondrial and midabdominal regions. The lower three-fifths, the ileum, lies in the right iliac fossa and pelvis. The jejunum can be distinguished from the ileum by its wider, thicker walls. The closely plicated mucous membrane and numerous villi in this region give the bowels a velvety texture. The jejunum is narrower and its walls are pale, membranous, and supple. Ovoid patches of lymphoid tissue, called Peyer's patches, occur along the free border of the ileum in children and young persons.

The mesentery connects the small intestine to the posterior abdominal wall along a line six inches long extending from the left side of the second lumbar vertebra to the right sacroiliac joint. It is disposed in numerous folds and is much thicker at its base than at the intestinal border. This thickening is the result of abundant fatty tissue, which increases in amount toward the distal end of the mesentery. At the jejunal end of the mesentery, the fat tissue ends about 1½ inches short of the gut wall, leaving translucent "windows." The ileal mesentery is

rendered completely opaque by the fatty tissue, which fills it and extends onto the gut wall.

The small intestine is supplied with blood by 12 or more jejunal and ileal branches of the superior mesenteric artery. These main branches form loops which give off small secondary and tertiary arcades forming an arterial network which increases in complexity toward the distal end of the intestine. Each terminal loop gives off vessels, the vasa recta, which pass to the gut wall where they supply single segments of intestine. If the vasa recta are interrupted, the intestinal wall becomes dependent on the precarious capillary anastomosis in the submucosa of the intestine.

Intestinal motility is an intrinsic property of the smooth muscle of the intestine. Movements are coordinated by the myenteric and submucosal ganglionic plexuses. Vagal fibers terminating in these plexuses intensify intestinal movements, whereas sympathetic fibers from the celiac plexus pass together with the blood vessels, serving to inhibit motility and regulate the blood supply. Sensation is carried by afferent fibers arising in the serosa and submucosa of the intestine, which is insensitive to all stimuli except spasm and dilatation.

The movements of the small intestine are usually classified into four groups. Pendular which is a rhythmic lengthening and contraction of the tube. Rhythmic segmentation is a sudden contraction of the intestine occurring 15 to 20 times a minute, subdividing the food many times. This occurs if all extrinsic nerves are cut and persists after application of cocaine and nicotine, so is myogenic in character. Peristalsis is a contraction above the bolus and a relaxation below, so that the food is gradually propelled aborally. The movements are dependent on the integrity of the myenteric plexuses. Roll movements are vigorous, swift waves that traverse the whole intestine. It is a mechanism for ridding the bowel of noxious substances and is also seen in asphyxia.

CONGENITAL ANOMALIES OF THE SMALL INTESTINE

Malrotation and nonrotation have been discussed above. Atresia where there is no lumen and stenosis where the lumen is narrowed are the result of arrested development of the embryonic intestinal tract. Although they may occur at any level, the commonest site is in the ileum. The symptoms are those of intestinal obstruction, the severity depending on the level of the affected bowel. If high, there may be little distention but early vomiting. If lower down, there is pain, restlessness, distention, visible peristalsis, and absence of the passage of normal meconium. Signs of dehydration soon appear. X-ray of the abdomen will often indicate the level of the lesion, the loops of intestine above being full of gas while the empty loops below cast no shadow.

Treatment.—The infant should be properly prepared with fluids and the affected loops short-circuited if possible. If a large portion of the bowel is involved, the case is hopeless. The postoperative course must be carefully followed, and the fluid, mineral, and food balance constantly watched.

Duplication of the Intestine

This anomaly is described below and may vary from a cyst of almost any size, containing all the coats of the bowel, to a completely differentiated portion of intestine, lying parallel to the original with the same mesentery and connected with it.

Meckel's Diverticulum

This anomaly, occurring in about 2% of the population, is due to persistence of the intestinal end of the vitello-intestinal duct. It is situated about 50 cm from the ileocecal valve on the antimesenteric border of the ileum. It may vary in size from a shallow out-pouching to a large tube extending to the abdominal wall. The obliteration of the vitello-intestinal duct may end at any level, thus accounting for the variations in size and shape. It may remain as a fibrous cord,

either attached to the anterior abdominal wall or floating free in the abdominal cavity. Many of these diverticula contain aberrant gastric mucosa, producing peptic ulceration in neighboring ileal mucosa. These ulcers frequently bleed and may perforate with resulting general peritonitis.

rant, is suggestive. X-rays with barium very rarely demonstrate the anomaly. In all laparotomies the ileum should be explored where possible.

Treatment.—Most patients are operated upon because of some complication of the diverticulum such as obstruction, intussus-

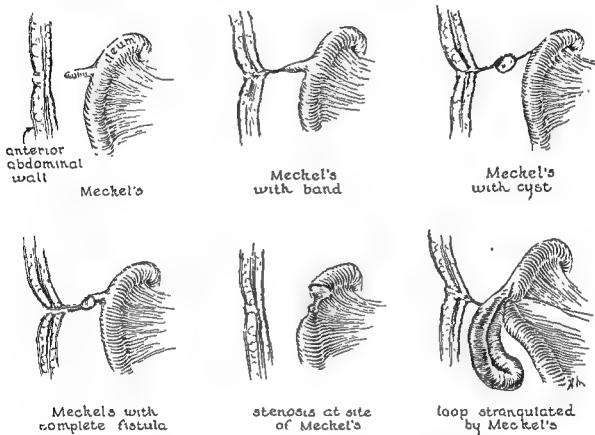


Fig 168—Varieties of Meckel's diverticulum.

Meckel's diverticula may become inflamed due to retained fecal material. The resulting edema prevents drainage, producing the picture of closed loop obstruction, with subsequent distention and perforation. The diverticulum may form the head of an intussusception. The attached or free end of the obliterated vitelline arteries may cause intestinal obstruction either as a compression band or a fulcrum around which a volvulus may occur.

Diagnosis is usually difficult, but melena occurring in a young adult especially, if associated with pain in the right lower quad-

ception, or perforation, and the diagnosis is seldom made before exploration. The urgent lesion must be relieved and the portion of ileum with the affected diverticulum resected. In uncomplicated cases, simple excision of the sac with inversion of the base is sufficient.

INFECTIONS OF THE SMALL INTESTINE

Acute infections of the small bowel are remarkably rare and are usually the result of injury, distention from obstruction, or

systemic disease. Enteritis, so-called, is often the result of chemical irritation and not, at the beginning at least, due to infecting organisms. Typhoid fever affects the lymph follicles, and perforations may occur requiring immediate surgical closure. Other organisms of the salmonella group may cause necrosis in the jejunum and are occasionally followed by stenosis.

Tuberculosis of the Intestine

Tuberculosis of the intestinal tract is almost always a complication of pulmonary tuberculosis, and may be present before the lung lesion has been detected. It is apparently caused by swallowed sputum, although hematogenous infection may play some part.

Pathology.—The commonest site is the lymphoid tissue of the ileocecal region, but the lesions may be found in isolated areas of the small or large bowel. It is usually an ulcerative lesion, but hyperplastic and sclerotic varieties occur. Microscopically there is the usual picture of caseation, with lymphocytes and giant cell formation.

Symptoms.—Symptoms are often complicated by the pulmonary picture, but abdominal cramps, diarrhea, and irregular fever are suspicious, especially if found during the course of treatment of pulmonary tuberculosis. In the more advanced stage, a mass may be present with signs of chronic or intermittent obstruction. Perforation with generalized peritonitis or localized abscesses may occur.

Diagnosis.—X-ray studies following a barium meal show hypermotility and failure of the cecum and ascending colon to retain the barium. There are occasionally other irregularities such as spasm and filling defects that can be demonstrated by proper radiologic techniques.

Treatment.—*Conservative*.—Early cases without stenosis should be given a trial on bed rest, high protein and carbohydrate diet, with supplemental feedings when necessary.

Streptomycin and other antibiotics and chemicals have given encouraging results.

Surgical.—This is sometimes a difficult decision and should be avoided in the acute phase if possible. It is indicated in perforation, where stenosis causes intermittent obstruction, in cases with fistula or abscess formation, and where the lesion is localized to a small segment of the bowel. Some patients are apparently improved by a simple laparotomy, but this is difficult to evaluate. Resection of the involved portion of the bowel is the operation of choice, but if this is impossible, a short-circuiting procedure should be performed.

Regional Ileitis (Chronic Stenosing Regional Enteritis, Crohn's Disease)

Regional ileitis, first described as an entity by Crohn, Ginzberg, and Oppenheimer in 1932, is a chronic, granulomatous lesion affecting principally the terminal ileum. It may, however, involve other areas of the small bowel and extend into the colon.

Etiology.—The infecting agent is unknown. It is commonest in early adult life, in the poorer economic group, and affects the sexes equally. Only a small number of cases have been reported in Negroes.

Pathology.—The diagnosis is often first made at operation. The terminal ileum in a typical case appears rigid, thickened, and red. Occasionally the diseased area is covered by fat or yellowish white exudate. Frequently it appears to have begun close to the ileocecal junction and extended proximally. Occasionally it involves the cecum and colon for varying distances. The mesentery is thick and edematous, and the lymph nodes are much enlarged. The inflammatory process may ulcerate through all coats of the bowel, forming abscesses that become localized by the matting together of coils of intestine, or forming internal fistulas between the adherent loops.

External fistulas through the abdominal wall are frequently present discharging pus

or feces. On section the bowel wall is thickened in all its coats. The lumen is narrowed and the mucosa hypertrophied and frequently ulcerated. Microscopically there are lymphatic hyperplasia, obstructed lymphatics, and giant cell formation. The disease occasionally involves several portions of the intestine with apparently healthy bowel between. These are often referred to as "skip areas."

Symptoms.—The symptoms are due to the inflammatory reaction, or stenosis, and thus resemble appendicitis, intestinal obstruction, or ulcerative colitis. Crampy abdominal pain localized in the right lower quadrant may be the first symptom, and the patient is operated upon for appendicitis. The disease is then recognized by the appearance of the bowel. Diarrhea, nutritional disturbances, anemia, and low-grade fever are common, and a mass may sometimes be palpated in the lower abdomen. The pain is inconstant and not related to meals. External fistulas occasionally follow appendectomy.

Diagnosis is made on the symptoms, course of the disease, and the x-ray appearance. A barium enema will exclude a lesion of the large bowel such as carcinoma or diverticulitis, and a barium drink followed by x-ray will often demonstrate the narrowed, rigid ileum.

Treatment.—An expectant approach may be taken when the acute process is found at laparotomy performed on the diagnosis of appendicitis. Some cases apparently are arrested without the development of obstruction, abscesses, or fistulas. Excision of the affected portion of the bowel is the best treatment. If there is too much involvement of the mesentery, a short-circuiting operation may be done, such as an ileo-transverse colostomy. The involved intestine may be removed later. The prognosis must be guarded as fistulas sometimes occur after any operative procedure, toxic signs may be present after a short-circuiting operation and recurrence may take place, even in apparently healthy bowel.

TUMORS OF THE SMALL INTESTINE

Tumors of the small bowel are remarkably rare. The commonest benign growths are adenomas which may be single or multiple; fibromas, which may arise in any layer; and lipomas, which may be single or multiple and may be pedunculated. Leiomyoma forms a mass projecting into the lumen of the bowel or into the peritoneal cavity. Sarcomatous changes occasionally occur in the growth. Pancreatic rests usually occur as small patches situated anywhere in the intestinal mucosa. They too may undergo malignant changes.

Symptoms.—The symptoms of tumors of the small intestine are usually due to complications such as obstruction, hemorrhage, or intussusception.

Treatment.—When discovered, small tumors may be removed by excision, but if this is impossible due to their size, resection of the involved segment of the intestine is indicated.

Malignant Tumors

Malignant tumors are adenocarcinomas and sarcomas. The adenocarcinoma may arise *de novo*, or from degeneration of a benign tumor or pancreatic rest. They may cause obstruction, melena, or an annular stricture. Metastases usually occur early.

Treatment.—The treatment is wide excision of the involved area with as much mesentery as possible. If metastases are present, the prognosis is poor. Short-circuiting procedures may be useful in relieving obstruction in cases not suitable for operation.

Sarcoma.—These develop as leiomyosarcomas, round cell sarcomas, and lymphoid tumors. They frequently grow quite large and metastasize early. The treatment is wide excision and the prognosis is bad.

Carcinoid.—These tumors arise from the argentaffine cells and occur most frequently in the submucosa of the appendix and termi-

nal ileum. They may be single or multiple solid nodules which have a golden yellow appearance on section. They may grow to an appreciable size and cause obstruction. A certain number, which some authors place as high as 20%, metastasize to the lymph glands and liver.

Mesenteric Lymphadenitis

Mesenteric lymphadenitis, which usually occurs in children, is characterized by attacks of crampy abdominal pain, nausea, occasional vomiting, and moderate elevation of temperature and white blood cell count. It is usually diagnosed as appendicitis. At operation the mesenteric lymph glands are markedly enlarged, reddened, and succulent, and free fluid is often present in the peritoneal cavity. The glands if removed at operation show only catarrhal inflammation on section, and cultures are sterile. Suppuration is rarely present. The etiology is obscure but it is probably due to a virus. It is frequently associated with lymphadenopathy elsewhere in the body. There is no evidence that it is due to trauma, tuberculosis, parasites, or enteritis.

Diagnosis can usually be made on a history of attacks of colicky pain and tenderness not characteristically situated over the appendix region. The tender area will sometimes shift to the midline when the patient lies on the left side due to the falling of the mesentery toward the left (Klein's test).

Treatment.—The attacks tend to disappear as the child grows older. The difficulty, however, is in excluding appendicitis with certainty, and the anxiety of the parents frequently leads to appendectomy. It is remarkable that many patients are completely relieved and others have fewer attacks. While mesenteric lymphadenitis is apparently a self-limited disease, appendectomy appears to have a beneficial effect on the course of the disease.

MESENTERIC VASCULAR OCCLUSION—MESENTERIC THROMBOSIS

The mesenteric veins may be the site of thrombosis occurring in association with cirrhosis of the liver, following splenectomy for some blood dyscrasias, external compression from tumors, and extension of thrombophlebitic processes in the ileocolic or hemorrhoidal veins. The mesenteric arteries, usually the superior, may be occluded by an embolus arising from the mitral valve in endocarditis, the left auricle, the pulmonary veins, or arteriosclerotic plaques from one of the major vessels.

Pathology.—In thrombosis the bowel becomes congested, swollen, and cyanosed. There may be a temporary diarrhea due to anoxia, but the bowel soon becomes paralyzed and gangrene is the usual outcome. However, collateral venous drainage may be established and the patient survives. In embolism the extent of the infarction depends on the site of the obstruction. Small areas may be saved by collateral channels, but if the superior mesenteric artery is involved, the whole small bowel and the proximal half of the colon may die. Occasionally circulation is re-established, and patients regarded as hopeless at operation have recovered.

Clinical Picture.—The condition usually appears in middle age, in a debilitated patient, following operation, or suffering from some other lesion as mentioned above. There are signs of an abdominal catastrophe often with agonizing pain and collapse. There may be vomiting of blood and melena. Death may quickly ensue or follow soon after from obstruction or peritonitis.

Treatment.—The patient must be treated by transfusions and made ready for operation. The involved bowel must then be resected if possible. It is remarkable that many patients have survived with only a small segment of intestine remaining and lived a reasonably normal life, so that the

operator need not despair if huge segments of bowel have to be removed. It is possible in cases of embolism, that the obstruction may be removed from the vessel, and circulation may be restored.

REFERENCES

- Alvarez, W. C.: Sixty Years of Vagotomy; A Review of Some 200 Articles, *Gastroenterology* 10: 413-441, 1948
- Appleby, L. H.: Prolapsing Gastric Mucosa, *J. Internat. Coll. Surgeons* 10: 135-142, 1947.
- Beaumont, W.: Experiments and Observations on the Gastric Juice and the Physiology of Digestion. Facsimile of the Original Edition of 1833, XIII International Physiological Congress, Boston, 1929
- Crile, G., Jr., and Brown, G. M., Jr.: Vagotomy as a Treatment for Marginal Ulcer, *Gastroenterology* 17: 14-20, 1951.
- Crohn, B. B.: Regional Ileitis, *Surg., Gynec. & Obst.* 68: 314, Feb., 1939.
- Dragstedt, L. R., Harper, P. V., Tovee, E. B., and Woodward, E. R.: Section of the Vagus Nerves to the Stomach in the Treatment of Peptic Ulcer, *Ann Surg* 126: 687-699, 1947.
- Grimson, K. S., et al.: Vagotomy. *Surgery* 27: 49-61, 1950.
- Jay, G. D., III, et al.: Meckel's Diverticulum, *Arch Surg* 61: 158-167, 1950
- Lewisohn, E. F.: Bleeding Peptic Ulcer, *Arch Surg* 59: 37-56, 1949.
- Loc, R. H.: Massive Hemorrhage in the Upper Part of the Gastrointestinal Tract, *Arch. Surg* 61: 183-192, 1950
- Marshall, S. F.: Regional Ileitis *New England J Med* 222: 375-382, 1940.
- Mayo, H. W., Jr.: The Physiological Basis of Operations for Duodenal, Gastric and Gastrojejunal Ulcer, St. Louis, 1949, The C. V. Mosby Company
- Miller, G. G.: Subtotal Gastrectomy for Gastro-duodenal Ulcer, *Canad. M. A. J.* 44: 570-575, 1941.
- Pack, G. T., and McNeer, G.: Total Gastrectomy for Cancer, A Collective Review of the Literature and Original Report of Twenty Cases, *Internat. Abstr. Surg.* 77: 265-299, 1943.
- Pavlov, I. P.: The Work of the Digestive Glands, ed 2, London, 1910, Chas. Griffin and Co.
- Poer, D. H.: Lymphosarcoma of the Gastrointestinal Tract, *Surgery* 23: 354-362, 1948.
- Potts, W. J.: Congenital Atresia of Intestine and Colon, *Surg., Gynec. & Obst.* 85: 14-19, 1947.
- Ransom, H. K.: Treatment of Jejunal Ulcer, *Arch. Surg.* 58: 684-700, 1949.
- Ripstein, C. R.: Duplication of the Small Intestine *Am J. Surg.* 78: 847-852, 1949.
- Schindler, R.: Relative Surgical Curability of Certain Gross Types of Gastric Carcinoma, *Surg., Gynec. & Obst* 83: 453-461, 1946
- State, D., et al.: Early Diagnosis of Gastric Cancer, *J. A. M. A.* 142: 1128-1132, 1950.
- Sullens, W. E., Steigmann, F., and Meyer, K. A.: Surgical Considerations in Hemorrhage of the Upper Part of the Gastrointestinal Tract, *Arch. Surg.* 59: 1244-1260, 1949.
- Thompson, H. L., and Oyster, J. M.: Neoplasms of the Stomach Other Than Carcinoma, *Gastroenterology* 15: 185-243, 1950.
- Trimble, I. R., and Lynn, D. H.: The Surgical Treatment of Duodenal, Gastric, and Anatomic Ulcer With Special Reference to Vagus Resection, *Surg., Gynec. & Obst* 90: 105-133, 1950.
- Wangenstein, O. H., and Lannin, B.: Criteria of an Acceptable Operation for Ulcer. The Importance of the Acid Factor, *Arch Surg* 44: 489-500, 1942.
- Wener, J., and Hoff, H. E.: The Neuro-humoral Aspects of Peptic Ulcer Formation, *Canad M. A. J.* 59: 115-140, 1948
- Wolf, S., and Wolff, H. G.: Human Gastric Function—An Experimental Study of a Man and His Stomach, New York, London, and Toronto, 1943, Oxford University Press.

nal ileum. They may be single or multiple solid nodules which have a golden yellow appearance on section. They may grow to an appreciable size and cause obstruction. A certain number, which some authors place as high as 20%, metastasize to the lymph glands and liver.

Mesenteric Lymphadenitis

Mesenteric lymphadenitis, which usually occurs in children, is characterized by attacks of crampy abdominal pain, nausea, occasional vomiting, and moderate elevation of temperature and white blood cell count. It is usually diagnosed as appendicitis. At operation the mesenteric lymph glands are markedly enlarged, reddened, and succulent, and free fluid is often present in the peritoneal cavity. The glands if removed at operation show only catarrhal inflammation on section, and cultures are sterile. Suppuration is rarely present. The etiology is obscure but it is probably due to a virus. It is frequently associated with lymphadenopathy elsewhere in the body. There is no evidence that it is due to trauma, tuberculosis, parasites, or enteritis.

Diagnosis can usually be made on a history of attacks of colicky pain and tenderness not characteristically situated over the appendix region. The tender area will sometimes shift to the midline when the patient lies on the left side due to the falling of the mesentery toward the left. (Klein's test.)

Treatment.—The attacks tend to disappear as the child grows older. The difficulty, however, is in excluding appendicitis with certainty, and the anxiety of the parents frequently leads to appendectomy. It is remarkable that many patients are completely relieved and others have fewer attacks. While mesenteric lymphadenitis is apparently a self-limited disease, appendectomy appears to have a beneficial effect on the course of the disease.

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Pylephlebitic Abscess

Etiology.—Any suppurative process within the abdomen may cause a septic thrombophlebitis in the portal vein. The appendix is the commonest site of origin. The infected thrombus extends along the vein to the liver, or emboli are set free, which are carried to the liver by the portal system.

Pathology.—The abscesses are usually multiple, vary greatly in size, and are more common in the right lobe. If the abscess is situated close to the surface of the liver, it may rupture into the peritoneal or pleural cavity.

Clinical Features.—The antecedent suppurative process is followed after an interval by the secondary development of septicemia with severe chills and fever. Eventually hepatic involvement occurs with right upper quadrant pain, an enlarged tender liver, and jaundice. If the splenic vein is involved, splenomegaly may occur.

Signs of intrathoracic disease indicated by a raised right diaphragm on x-ray examination and signs of pleural involvement are common.

Treatment.—The early treatment of intra-abdominal suppuration by adequate surgery and appropriate antibiotic therapy will usually prevent pylephlebitis. If the laparotomy reveals evidence of thrombosis of the portal system, the involved veins may be ligated to prevent proximal spread and a course of anticoagulant therapy should be given.

If liver abscesses develop, they should be drained preferably by an extraperitoneal approach. Anticoagulants and antibiotic therapy should be given in maximum dosage.

Cholangitic Abscess

These abscesses form secondary to acute suppurative cholangitis and hence occur along the intrahepatic bile ducts. The clinical picture is one of intermittent fever, chills, jaundice, and a tender enlarged liver.

The treatment is drainage of the common duct and antibiotic therapy. If any large abscesses are present, they should be drained extraperitoneally.

Amebic Abscess

Etiology.—The *Entamoeba histolytica*, found in many parts of the world, may cause a hepatitis which in some cases goes on to abscess formation.

Pathology.—Amebic abscesses are usually solitary and occur or develop in the right lobe of the liver. The amebae reach the liver via the portal circulation from the primary intestinal ulcers.

The abscess is formed by liquefaction of an area of hepatitis by a proteolytic toxin produced by the amebae. The abscess is typically sterile, and only by scraping the wall can amebae be demonstrated.

Clinical Picture.—An abscess may develop at any stage of the disease. The symptoms are fever, profuse sweating, weakness, and marked gastrointestinal upset. There is pain in the right upper quadrant and an enlarged tender liver. X-ray shows an elevation and immobilization of the right diaphragm. The presence of the *Entamoeba histolytica* in the stool confirms the diagnosis.

Treatment.—Emetine is a specific agent for the intestinal ulceration and hepatitis. When abscesses form, drainage of the abscess is essential. Every precaution must be taken to maintain the sterility of the abscess, and this may best be done by repeated aspirations.

ACTINOMYCOSIS

Actinomycosis of the liver is very rare and develops from a focus of infection in the abdomen or thorax (most commonly in the ileocecal region).

Pathology.—The liver is enlarged and adherent to surrounding structures. It is pitted with numerous small abscesses giving it a honey-combed appearance. The disease may invade the abdominal wall with resulting sinus formation.

CHAPTER XIX

LIVER

J. F. HOPKIRK, M.D., AND R. C. LONG, M.D

ANATOMY AND PHYSIOLOGY

Anatomy.—The liver is the largest glandular organ in the body. It is wedge-shaped and situated in the right upper part of the abdominal cavity. The larger right lobe is in close relationship to the diaphragm which intervenes between it and the right lung and pleura, while the smaller left lobe extends to the left subdiaphragmatic region.

Blood Supply.—The liver obtains its blood supply from two sources, the portal vein and the hepatic artery. The venous return is by a single system, the hepatic vein which drains directly into the inferior vena cava.

Physiology.—The functions of the liver are many and complex and can be listed as follows:

1 Excretory: i.e., formation and excretion of bile (see section on Jaundice)

2 Metabolic. The liver plays an important role in metabolism (e.g., glycogen storage)

3 Detoxification. Certain poisonous substances which reach the liver via the portal circulation are detoxified by the liver and reticulo-endothelial cells (Kupffer)

4 Hematopoietic: The liver is concerned in the storage of the antipernicious anemia factor, iron and copper

5 Blood coagulation: i.e., production of fibrinogen and prothrombin

6 Heat production: The liver and the muscles, as the result of metabolic processes occurring in them, are the main sources of body heat

INJURIES TO THE LIVER

Etiology.—Injury may occur as the result of blunt trauma to the right upper abdomen or lower thorax or from penetrating wounds

Pathology.—The right lobe is usually involved. The degree of damage varies from simple contusion to subcapsular or even complete rupture. Associated injury of the abdominal and thoracic viscera commonly occurs

Clinical Picture.—In the absence of an external wound, there may be some difficulty in making the diagnosis. Abdominal pain with tenderness in the right hypochondrium is characteristic. If the tear is large, signs of hemorrhagic shock appear and dullness in the flanks may result from the blood in the peritoneal cavity. An x-ray of the chest and abdomen should always be taken to help rule out injury to other viscera.

Treatment.—In the absence of injury to other abdominal viscera, the treatment should be conservative. Whole blood transfusions may be necessary to restore the blood volume and overcome shock. Uncontrolled hemorrhage requires laparotomy and suture of the liver tear

INFECTIONS OF THE LIVER

Liver Abscess

Etiology.—Abscesses develop from infection which reaches the liver from the systemic circulation in general pyemia, from the portal vein in septic thrombophlebitis, from the bile duct in acute cholangitis or from a neighboring focus by direct spread.

Pyemic Abscess

Etiology.—In any case of general pyemia, multiple septic emboli are set free, some of which reach the liver via the hepatic artery causing numerous small abscesses. These are seldom suspected during life unless jaundice occurs. The treatment is that of the general systemic infection

Benign Tumors

Benign tumors are an uncommon cause of symptoms although they are frequently found at operation and postmortem. Adenomas and hemangiomas may become large enough to present as a mass in the upper abdomen or may press upon neighboring viscera to cause symptoms.

The treatment of these tumors should be excision if possible.

Malignant Tumors

1. PRIMARY CARCINOMA

Primary carcinoma of the liver is a rare disease. It arises from either the liver cells (hepatoma) or from the cells of bile ducts (cholangioma). The *hepatoma* is commoner in the male and is frequently preceded by portal cirrhosis. The *cholangioma* is commoner in females and this fact may have some relationship to the greater incidence of gall bladder disease in this sex.

Pathology.—The tumor may take the form of multiple nodules, a single large mass, or a diffusely infiltrating growth. In *hepatoma* the cells are arranged in cords or solid alveoli, and contain bile pigment. *Cholangiomas* consist of closely packed tubules with a highly vascular stroma. Metastases to other organs are rare.

Clinical Picture.—The onset is insidious with anorexia, loss of weight, and anemia. An enlarged liver, emaciation, ascites, and perhaps jaundice make the diagnosis more obvious. The disease runs a rapid and downhill course with death occurring within three or four months after the onset of symptoms. Liver biopsy is necessary to establish a correct pathological diagnosis.

Treatment.—In the diffuse form or where multiple nodules are present, nothing can be done. Rarely, when a solitary primary tumor is found, resection of a part or all of a lobe may be undertaken. X-ray therapy is without effect.

2. PRIMARY SARCOMA

Primary sarcoma is an extremely rare lesion. The treatment is one of palliation although some temporary relief may be expected from x-ray therapy.

3. SECONDARY CARCINOMA

Secondary carcinoma of the liver is very common in patients with malignant disease. The primary growth may lie in any part of the gastrointestinal tract drained by the portal vein, it may arise elsewhere in the body, e.g., uterus, bronchus, breast, and reach the liver by the systemic circulation or it may invade the liver by direct extension or by the lymphatics.

The metastases are nearly always multiple. The liver progressively enlarges, and presents a nodular surface. Pain, jaundice, and ascites eventually occur.

Treatment.—Treatment should be directed toward the alleviation of symptoms. However, in the rare case where the primary growth has been adequately excised and a solitary metastasis develops in the liver, resection of the involved lobe may be advisable.

SURGICAL JAUNDICE

Jaundice is a manifestation of an increase in the bilirubin level of the blood which gives a yellow discoloration of the skin, sclerae and mucous membranes. The hyperbilirubinemia results from a number of diverse mechanisms, not all of which are associated with the liver and biliary system.

Pathogenesis of Jaundice.—When the red blood cell is broken down, it divides into an iron-free molecule and an iron-containing portion (hemosiderin). It is the iron-free molecule which is the precursor of bilirubin. The cells of the reticulo-endothelial system convert this molecule to bilirubin-globin which is removed from the blood stream by the liver and is excreted as sodium bilirubin-ate by the liver cells as one of the components of the bile. In the gastrointestinal

Clinical Picture.—The initial symptoms are those of the primary disease. Upper abdominal pain, fever, and an enlarged tender liver are signs of hepatic involvement.

Treatment.—Antibiotic therapy will arrest the disease in many instances. (See Bacteriology, page 42) If the abscess is accessible, it should be drained and the necrotic tissue excised.

CYSTS OF THE LIVER

Retention Cysts

Retention cysts are small single cysts, lined with a flattened epithelium and are thought to be due to a malformation of the smaller bile ducts. They are usually asymptomatic unless large enough to press upon surrounding structures.

Treatment.—The cyst should be excised if symptoms develop.

Polycystic Disease

Polycystic disease frequently occurs in association with polycystic kidneys. The cysts are multiple, vary greatly in size and contain a clear albuminous fluid.

Treatment.—Treatment is usually unsatisfactory because of the wide distribution of the cysts in the liver and in other organs.

Parasitic Cysts (Hydatid Disease)

Etiology.—The *Taenia echinococcus* is ingested by man in food contaminated by the excreta of dogs and other canines. The embryos penetrate the intestinal wall and are carried to the liver by the portal vein. The great majority of the embryos are arrested in the liver but may pass through into the systemic circulation and lodge in the lungs or other organs.

Pathology.—When the embryo reaches the liver, it becomes encysted and the cyst gradually increases in size. The fully developed cyst has two layers, an outer laminated fibrous layer and an inner germinal layer from whose cells grow daughter cysts. The parent cyst is usually solitary and con-

tains a clear gelatinous material in which are imbedded many daughter or grand-daughter cysts.

Clinical Picture.—When the cysts are small, they are asymptomatic; but as the disease progresses, the cysts enlarge and symptoms develop. Dull pain in the right hypochondrium is common and jaundice may occur. Physical examination will usually reveal a palpable mass on the surface of the liver.

Urticaria, eosinophilia, and other manifestations of the developing allergic phenomena are seen. As calcification of the cyst wall is common, a plain film of the abdomen may be of help in the diagnosis.

There are two confirmatory tests.

1 The Casoni reaction—the intradermal injection of the fresh fluid from animal hydatid cysts.

2 The Chedini-Weinberg complement-fixation reaction.

The cysts may rupture into

1 The peritoneal cavity,

2 The pleural cavity, lung or bronchus with the development of a bronchobiliary fistula, or

3. The gastrointestinal tract.

Treatment.—Operation is the treatment of choice. Careful localization of the cyst is essential in order that the incision may be placed correctly. When the cyst is exposed and carefully packed off, it is aspirated and the contents are replaced by a solution of 10% formalin. The cyst is opened and the inner layers are carefully excised. The cavity is closed and the abdomen is sutured without drainage. Partial hepatectomy may be required if the cysts are multiple.

TUMORS OF THE LIVER

Benign.—Adenoma, lymphangioma, hemangioma, myoma, teratoma, fibroma.

Malignant.—1 Primary carcinoma (hepatoma, cholangioma), sarcoma.

2. Secondary.

even disappear for a time. It is usually accompanied by pruritus which is due to the retained bile salts. This symptom does not occur as frequently in the nonobstructive types of jaundice. On physical examination

color with the fluctuations in the degree of icterus. The urine is bile colored.

2. *Malignant obstruction of the common duct.* The jaundice develops slowly and progressively and is not typically associated

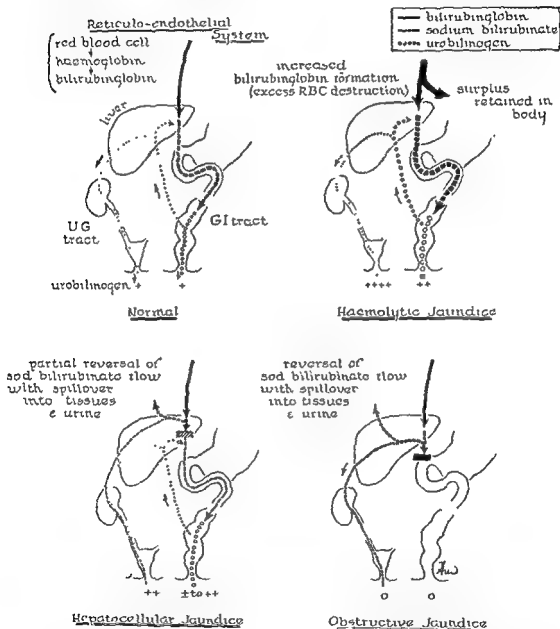


Fig 189.—Diagrammatic representation of normal and abnormal bile metabolism. The size of the arrows shows in an approximate fashion the magnitude of abnormal bile or bile product flow.

there may be tenderness over the gall bladder but the jaundice is the only other physical sign of value. The stools are clay colored at the height of the jaundice, but vary in

with attacks of biliary colic although dull upper abdominal pain is frequent. There are commonly symptoms of anorexia, loss of weight, and a general lack of well-being. If

tract the bilirubin is acted upon by bacteria and is reduced to urobilinogen most of which is excreted in the stool. The remainder is reabsorbed and carried by the portal circulation to the liver. Here the greater part is reconverted to bilirubin, but some reaches the systemic circulation, and is excreted in the urine. The unabsorbed urobilinogen in the bowel is oxidized to urobilin which is partly responsible for the color of normal stools.

Jaundice is due either to an increased rate of bilirubin production or to a decrease in the rate of its excretion. An increased rate of production may be due to:

1 Increased susceptibility of the red blood cells to hemolysis, e.g., congenital hemolytic jaundice

2. Presence of hemolysin, e.g., incompatible blood transfusions.

3 An overactive reticulo-endothelial system.

A decreased rate of excretion may be due to:

(1) damage to liver parenchymal cells
(a) in infections, e.g., infectious hepatitis

(b) toxic agents, e.g., phosphorus, arsenic

(c) in cirrhosis,

(2) obstruction to the flow of bile which is usually extrahepatic, e.g., stone or tumor

Classification of Jaundice.—There are three main types, however, it is difficult to differentiate these exactly, as there is frequently an overlap of one type with another

1 Hemolytic due to the excessive production of bilirubin from the destruction of large numbers of erythrocytes, e.g., congenital hemolytic jaundice.

2 Hepatocellular due to a disturbance in the liver cells from

(a) infections due to virus infection, e.g., leptospiral infection, syphilis, etc.

(b) chemical poisons: alcohol, arsenic, chloroform, carbon tetrachloride,

(c) biological substances: incompatible blood,

(d) miscellaneous: portal cirrhosis

3. Obstructive due to some obstruction to the flow of bile.

(a) Within the lumen of the bile ducts, e.g., stone, inflammatory exudate (cholangitis), parasites (*Ascaris*).

(b) Changes in the wall of the duct: e.g., stricture (congenital, inflammatory, traumatic); inflammation, neoplasm, or spasm of the sphincter of Oddi.

(c) Pressure on the ducts from without: pancreatic lesions (neoplasm, cysts, pancreatitis), hepatic lesions (neoplasm, abscess, gumma), enlarged glands at portal fissure (carcinoma, tuberculosis, Hodgkin's disease); duodenal and gastric lesions (tumors, ulcers).

4. Mixed: when the initial lesion, e.g., obstruction, is followed by secondary changes in the liver cells which contribute to the duration and severity of the jaundice.

Differential Diagnosis of Jaundice.—There are many causes of jaundice but there are four types which most commonly confront the surgeon and which he must distinguish

1 Stone in the common duct.

2 Carcinoma of the pancreas

3 Hemolytic jaundice

4 Infectious hepatitis.

To reach a correct diagnosis a complete history and physical examination and special laboratory procedures must be used.

Clinical Picture.—Much can be learned and often a diagnosis can be made from an accurate clinical history:

1. *Jaundice due to common duct stone*

There is a long history of biliary dyspepsia, with recurrent attacks of biliary colic and the jaundice is commonly preceded by such an attack. The icterus is mild in degree, varies in depth from week to week, and may

Urobilinogen Content of Urine and Feces.

—(Normal: Urine, 0 to 3.5 mg per day. Feces, 40 to 280 mg. per day.) The estimation of the urobilinogen content of the urine and stool is of great value in the differential diagnosis of jaundice, and in evaluating the progress of the disease. In complete obstructive jaundice, where no bilirubin reaches the intestine, urobilinogen will be absent from both the feces and the urine. The stools are clay colored. In hepatocellular jaundice the ability of the liver to re-excrete the urobilinogen is impaired, the blood level rises, and the amount excreted in the urine is increased. In hemolytic jaundice, with consequent increase in erythrocyte destruction and thus an increased bilirubin formation, the level of the urobilinogen in stool and urine is markedly increased.

Bromsulphalein Test.—(Normal: 95% of dye excreted in 30 minutes.) The liver has the capacity to remove certain dyes from the circulation and excrete them in the bile. Liver disease prevents this excretion and a high percentage of the dye may be retained. As this is a colorimetric test, it is invalidated in the presence of jaundice, and because of the added burden placed upon the liver its use in cases suspected of having obstructive jaundice is contraindicated. Its chief value lies in the assessment of liver function in the patients without jaundice.

Albumin, globulin, fibrinogen, and prothrombin are formed in the liver. Changes in the metabolism of these protein fractions occur in liver disease.

1. Serum Protein.—(Normal: total 6.9 to 8.5 mg. per 100 ml; albumin 4.4 to 6.0 mg per 100 ml.; globulin 1.5 to 3.0 mg per 100 ml.) In advanced liver disease a marked decrease occurs in the total serum proteins, and this is chiefly, if not entirely, in the albumin fraction. Occasionally there may be a rise in the serum globulin level which leads to a reversal of the normal albumin-globulin ratio and even may result

in an increase in the level of the total serum protein level.

2. Takata-Ara Reaction.—Liver disease resulting in changes in the gamma globulin of the serum gives a flocculation reaction when the serum is combined with mercuric chloride. It is usually positive in cases of hepatocellular jaundice, particularly when associated with cirrhosis, but is negative in simple obstructive jaundice.

3. Prothrombin.—Prothrombin is formed by the liver in the presence of an adequate supply of vitamin K. Two factors are therefore necessary for its production—adequate intake and absorption of vitamin K and a functioning liver. In simple obstructive jaundice the exclusion of bile salts from the intestine and the consequent lack of absorption of the fat-soluble vitamin K result in a decreased formation of prothrombin. In such cases parenteral administration of vitamin K will bring the prothrombin concentration to normal. In severe hepatocellular damage there is also a decreased prothrombin formation, but this is refractory to the administration of vitamin K. This failure to respond occurs only in the presence of severe liver damage.

4. Cephalin-Cholesterol Flocculation Test.—In hepatocellular damage, gamma-globulin is produced in increased amount while serum albumin formation is decreased. This change in the serum proteins causes a flocculation of a cephalin-cholesterol emulsion. This test is unaffected by jaundice and is a very sensitive test of hepatic function. Its chief value lies in differentiating obstructive from hepatocellular jaundice. In hepatocellular jaundice strongly positive reactions (3 or 4 plus) are the rule. In liver damage due to long-standing biliary obstruction, the test is positive, but not usually more than 1 or 2 plus.

5. Thymol Turbidity Test and Thymol Flocculation Test.—(Normal: turbidity, less than 4 units; flocculation 0 to 1 plus.) This is a flocculation test similar to the cephalin-

the obstruction is below the entrance of the cystic duct, the gall bladder is enlarged and palpable (Courvoisier's Law). Occasionally ulceration of the tumor leads to temporary decrease in the jaundice and the presence of occult blood in the stools. The jaundice is accompanied by pruritus.

3 *Infectious Hepatitis* The jaundice follows a period of anorexia, nausea and vomiting, and is not associated with pain. Occasionally fever is present. The jaundice deepens rapidly, usually lasts for 3 to 4 weeks and then gradually fades. The liver is enlarged and tender.

4 *Hemolytic Jaundice* This may be congenital or acquired. The diagnosis is readily made by examination of the urine, stool, and blood. (See Spleen.)

Special Investigations

Liver Function Studies.—The diagnosis of the cause of the jaundice frequently presents great difficulty and certain laboratory procedures are used to aid in the differentiation. Once the diagnosis has been established, these tests may be utilized to follow the progress of the disease. No single test has been found that will measure accurately liver function, and it is therefore necessary to employ a group of procedures each of which measures a different aspect of liver physiology. If obstructive jaundice is of long standing, liver function studies are unreliable because of the secondary hepatocellular damage. While it is impossible to cover each of the tests in detail, the most useful will be considered.

CLASSIFICATION OF LIVER FUNCTION STUDIES

- 1 Tests based upon the metabolism of bile pigments.
 - (i) Quantitative serum bilirubin (van den Bergh).
 - (ii) Urobilinogen content of urine and feces.
- 2 Tests based upon the excretory function of the liver.
 - (i) Bromsulphalein

3. Tests based upon the activity of the liver in protein metabolism

- (i) Serum protein: total, albumin and globulin.
- (ii) Takata-Ara reaction
- (iii) Blood prothrombin time and its response to vitamin K.
- (iv) Cephalin-cholesterol flocculation test.
- (v) Thymol turbidity and thymol flocculation.

4 Tests based on the activity of the liver in lipid metabolism.

- (i) Total cholesterol level of the blood.
- (ii) Ratio of total cholesterol to cholesterol esters

5 Tests based upon the detoxifying function of the liver.

- (i) Hippuric acid synthesis

6 Miscellaneous.

- (i) Alkaline phosphatase level.

Quantitative Serum Bilirubin.—(Normal: 0.1 to 0.8 mg. per 100 ml.) Jaundice is best measured by the quantitative estimation of the serum bilirubin. A slight degree of hyperbilirubinemia may not cause visible discoloration of the tissues. However, jaundice becomes clinically evident when the serum bilirubin level reaches 2.5 to 3.0 mg. per 100 ml. The total bilirubin is made up of two fractions, the direct and the indirect. The *indirect van den Bergh* is the measure of bilirubin before it has passed through the liver cells (bilirubinglobin). It is so called because it is chemically inactive and will not combine with the reagent (Ehrlich's diazo reagent) without the addition of a substance which lowers its surface tension. Once it has passed through the liver cells, it is changed to sodium bilirubinate which readily reacts with the reagent, and gives the *direct reaction*. Normally 40 to 75% of the total bilirubin is of the direct variety. An *indirect reaction* is found in cases of hemolytic jaundice, while a direct reaction is found in both obstructive and hepatocellular jaundice.

CHAPTER XX

THE BILIARY SYSTEM

J. F. HOPKIRK, M.D., AND R. C. LONG, M.D.

The hepatic duct is formed in the depth of the transverse fissure of the liver by the union of the ducts draining the right and left lobes. It runs inferiorly and posteriorly in the edge of the lesser omentum, lying anterior and to the right of the portal vein. The hepatic artery usually lies a short distance from its left margin.

The gall bladder is a thin walled, muscolumembranous organ about 10 cm in length which lies in a fossa on the inferior surface of the right lobe of the liver. It is attached to the liver by loose connective tissue and is covered with peritoneum that is reflected from its sides onto the liver. The organ is divided into three parts: fundus, body and neck. The wide end or fundus usually reaches the anterior border of the liver and may come in contact with the anterior abdominal wall. If the gall bladder is distended, it may be palpable in the angle between the right rectus muscle and the costal margin. The body lies in close relation to the duodenum, pyloric end of the stomach, and transverse colon. The neck is an S-shaped tube which empties into the cystic duct. Usually there is a sacculation at the neck, known as Hartmann's pouch.

The cystic duct runs posteriorly and medially to join the hepatic duct.

The common bile duct is the direct continuation of the hepatic duct after its junction with the cystic duct. It passes inferiorly in the edge of the lesser omentum, then behind the first part of the duodenum to run in a groove in the head of the pancreas and enter the second part of the duodenum at the ampulla of Vater $2\frac{1}{2}$ cm. below the pylorus.

Blood Supply.—The common hepatic artery arises from the celiac axis, passes superiorly in the lesser omentum, and divides near the liver into the right and left hepatic arteries. The cystic artery usually arises from the right hepatic branch. It lies a short distance to the left of the cystic duct and supplies both the duct and the gall bladder. It should be emphasized that the course of the artery and its relation to the adjacent structures are subject to wide variation, and it is important therefore that all structures should be clearly visualized at operation.

Lymphatic Drainage.—Most of the lymphatic drainage converges on the cystic lymph gland which lies close to the junction of the cystic and common duct. Some of the lymphatics pass across the bare area of the gall bladder wall directly into the liver.

Nerve Supply.—The innervation of the gall bladder and bile ducts is through the splanchnics and the right branch of the vagus nerve.

Embryology.—During the fourth week of fetal life a solid budlike outpouching forms on the ventral wall of the primitive foregut from which develop the liver and the extrahepatic biliary system. This solid bud forms vacuoles which coalesce to form the gall bladder and ductal system. Any arrest of development at this stage may result in stricture or a congenital absence of the ducts or gall bladder. If, on the other hand, the bud splits in an abnormal manner, a double gall bladder, aberrant ducts, or diverticula of the gall bladder may result.

Physiology.—The principal function of the gall bladder is that of a reservoir for liver

cholesterol test but is considerably less sensitive. It too is unaffected by the presence of jaundice. In jaundice due to extrahepatic biliary obstruction, the values are within normal limits. In hepatocellular jaundice the values are elevated. The thymol flocculation test is a continuation of the thymol turbidity procedure, and in the absence of jaundice is much more sensitive.

6 Plasma Cholesterol and Cholesterol Esters.—(Normal: total cholesterol 150 to 250 mg per 100 ml; cholesterol esters, 75 to 175 mg. per 100 ml.) Hypercholesterolemia commonly occurs in obstructive jaundice, and the ratio of total cholesterol and cholesterol esters is normal. In very severe hepato-

duct. The renal function must be normal if this test is to be accurate.

8. Alkaline Phosphatase.—(Normal: 3 to 13 units [King-Armstrong]) Alkaline phosphatase is an enzyme produced by the osteoblasts at the site of bone formation. It is excreted by the liver in the bile. In obstructive jaundice the alkaline phosphatase is markedly increased but is normal or very slightly elevated in hepatocellular jaundice.

Liver Biopsy.—A direct examination of the liver parenchyma by biopsy with a hollow needle is of value, particularly in cases of long-standing jaundice when other tests are inconclusive. There is some danger of hemorrhage following this procedure.

TABLE VII
URINE AND STOOL BILE AND UROBILINOGEN LEVELS IN JAUNDICED PATIENTS

| | URINE | | FECES |
|-------------------------|-------|--------------|--------------|
| | Bile | Urobilinogen | Urobilinogen |
| Normal | 0 | + | + |
| Hemolytic jaundice | II | ++++ | ++ |
| Hepatocellular jaundice | + | 0 | + or ++ |
| Obstructive jaundice | ++ | 0 | 0 |

TABLE VIII
LIVER FUNCTION VALUES IN JAUNDICE

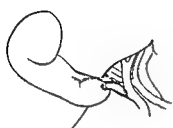
| NORMAL | HEMOLYTIC JAUNDICE | OBSTRUCTIVE JAUNDICE | HEPATOCELLULAR JAUNDICE |
|-------------------------|--------------------|----------------------|-------------------------|
| Bromsulphalein | not used | not used | not used |
| Serum protein | normal | normal | decreased |
| A/G ratio | normal | normal | reversed |
| Takata-Ara reaction | normal | normal | positive |
| Prothrombin | normal | decreased | decreased |
| Cephalin-cholesterol | normal | normal or 1 plus | 2 or more plus |
| Thymol turbidity | normal | normal | increased |
| Thymol flocculation | normal | 0 to 1 plus | over 1 plus |
| Cholesterol total | normal | increased | decreased |
| Cholesterol esters | normal | increased | decreased |
| Hippuric acid synthesis | normal | normal | decreased |
| Alkaline phosphatase | normal | increased | normal |

cellular damage, lowered values frequently occur, and the proportion of cholesterol esters is diminished.

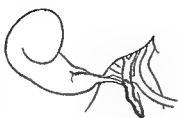
7. Hippuric Acid Synthesis.—(Normal: over 1 gram) Benzoic acid is conjugated in the liver with glycine to form hippuric acid which is excreted in the urine. The amount excreted is subnormal in patients with hepatocellular jaundice, but is normal in uncomplicated obstruction to the common

REFERENCES

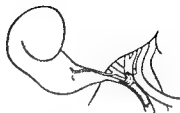
- Cantarow, A., and Trumper, M. Clinical Biochemistry, ed 4, Philadelphia, 1949, W. B. Saunders Company.
- Young, L. E. Current Concepts of Jaundice With Particular Reference to Hepatitis, New England J Med 237: 225-231 and 261-268, 1947.
- Gastroenterology, Philadelphia, 1949, W. B. Saunders Company, vol 3, pp 103-206.



short large cystic duct



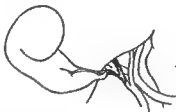
mobile common duct



cystic duct parallel to hepatic duct



accessory hepatic duct



cystic duct joins R hepatic duct



large, pendulous Hartmann's pouch

Fig 171—Variations in the position of the extrahepatic bile ducts. In operations in this area extra care must be taken to identify each structure prior to its ligation or section



R hepatic a. anterior to hepatic duct



R hepatic a. lying close to cystic duct



two cystic arteries

Fig 172—Variations in the position of right hepatic and cystic arteries. Ligation of the right hepatic artery may occur when anomalies are present. This accident, although no longer believed lethal, should never occur.

bile. Normally, almost the entire flow of bile from the liver (500 to 1,000 c.c. daily) is carried into the gall bladder, where it is concentrated as much as tenfold by absorption of water and inorganic salts. After food is eaten, there is a contraction of the gall bladder associated with a relaxation of the sphincter of Oddi. Evidence indicates that the emptying of the gall bladder is chiefly under hormonal control. Cholecystokinin is released by the mucosa of the duodenum and small intestine under the stimulus of food, and when absorbed into the blood, causes the contracture.

not concentrate radio-opaque dye that is excreted by the liver. In a relatively small number of patients several factors may influence the accuracy of the test:

(a) Failure to absorb the dye from the gastrointestinal tract, e.g., in vomiting or diarrhea.

(b) Failure of the liver to remove the dye from the blood stream and excrete it, e.g., in liver disease.

(c) Obstruction to the cystic duct.

(d) Premature emptying of the gall bladder before films are made, e.g., following ingestion of food prior to radiography.

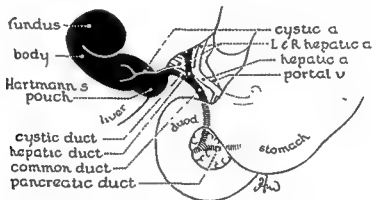


Fig 170.—Normal anatomy of the extrahepatic biliary system. Note how the intrapancreatic portion of the common duct can be occluded by disease in the head of the pancreas.

The chief components of the bile are bile pigments, the alkali salts of bile acids, inorganic salts, cholesterol, and mucoprotein.

Diagnostic Procedures

Simple Roentgenography.—Most gallstones are not shown on plain roentgenograms, as only those with a high calcium content cast a definite positive shadow. Cholesterol stones are not radio-opaque. Therefore, a negative roentgenogram is of little significance in excluding disease of the gall bladder.

Cholecystography.—The concentrating power of the gall bladder is of great clinical importance because the Graham-Cole test for cholecystitic disease is based on the assumption that a diseased gall bladder will

(e) Presence of other shadows in the right upper quadrant which interfere with interpretation of the gall bladder shadow.

(f) Failure of the gall bladder to concentrate the dye to an extent sufficient to produce a shadow

The contrast medium used is tetraiodophenol-phthalein (T.I.P.P. test) or iodoaliphonic acid (Priodex). This is preferably given by mouth. Intravenous injection of the dye may be necessary if cholecystography is to be done in a patient having pyloric obstruction or ulcerating lesions of the gastrointestinal tract. This test will give a positive diagnosis of cholelithiasis in a high percentage of cases. The accuracy of the test is considerably less in noncalculous dis-



Fig. 173.—Calcified gall bladder.



Fig. 174.—Cholecystograms. Normal gall bladder before and after a fatty meal with visualization of the cystic and common ducts.

blocks the outflow of bile and causes an accumulation of the normal secretions within the gall bladder. The resulting distention plus the edema at the site of obstruction impairs the arterial supply and the venous and lymphatic drainage of the gall bladder. The resulting ischemia leads to necrosis and ulceration, which may go on to gangrene and perforation.

An overconcentration of bile salts is capable of injuring normal gall bladder tissue. This may be an added etiological factor. Such an injured gall bladder wall is particularly liable to secondary bacterial invasion by *Streptococcus hemolyticus*.

Pathology.—The wall of the gall bladder is red, thickened, and edematous. The serosal surface is congested and is often covered with fibrin. The mucosa is bright red. If the obstruction to the cystic duct is complete, the gall bladder is distended with what appears to be purulent fluid—the so-called *empyema of the gall bladder*. This fluid may be a sterile emulsion of cholesterol, mucus, and calcium carbonate, but true pus may be present.

Edema is responsible for most of the thickening of the wall. The inflammatory exudate is most marked in the outer layers and is characterized by a relatively small number of polymorphonuclear cells. A hemorrhagic exudate is a prominent feature. Not infrequently there is an associated pancreatitis.

A considerable number of acutely inflamed gall bladders yield positive bacterial cultures. The common organisms found are *E. Coli*, streptococcus and staphylococcus and gram-negative bacilli.

Clinical Picture.—There is frequently a history suggestive of previous gall bladder disease. The onset of symptoms in the acute attack is usually sudden, with pain in the right hypochondrium reaching its peak in 24 hours. The pain may radiate to the right scapular region and to the right shoulder. Nausea and vomiting are common.

Tenderness over the gall bladder is constant and is associated with muscle spasm. The gall bladder is usually palpable despite this muscle resistance. Fever is common and is accompanied by tachycardia, and leukocytosis. Jaundice may occur occasionally if there is an associated hepatitis, cholangitis, or common duct obstruction due to stone, inflammatory edema or pressure on the common duct from swelling of Hartmann's pouch.

The acute symptoms usually subside in 2 to 3 days, but tenderness, rigidity, and a palpable mass are likely to persist for a considerable period. However, the improvement in the clinical picture is often misleading and cannot be directly correlated with a corresponding improvement in the disease process. The condition may go on to abscess formation and even gangrene.

Differential Diagnosis.—Acute cholecystitis must be differentiated from perforated or penetrating peptic ulcer, acute appendicitis, acute pancreatitis, and coronary thrombosis.

With perforated peptic ulcer, there is often a previous history of epigastric pain which was relieved by food and alkalis. The tenderness is acute and the rigidity is boardlike; both are more generalized, and there is usually a loss of liver dullness; an x-ray of the abdomen may show air in the peritoneal cavity. The differentiation from acute pancreatitis is difficult. The pain and tenderness in pancreatitis are more diffuse, tend to radiate to the left lumbar region, and vomiting is more severe. An elevated serum amylase is of diagnostic significance.

In acute appendicitis the location of maximum pain and tenderness is in the right iliac fossa or loin and is associated with rectal tenderness. In the high-lying retrocecal appendix the differentiation may be impossible. In coronary thrombosis the pain frequently radiates down the left arm. There is hypotension with typical electrocardiographic changes. However, similar changes in the electrocardiogram may result from an attack of acute cholecystitis.

case of the gall bladder. In normal gall bladders a false positive test may occur

Duodenal Drainage.—Duodenal intubation is of value as a diagnostic measure. A tube is passed into the duodenum and gall bladder contraction is stimulated by the introduction of magnesium sulphate or olive oil. The presence of crystals of cholesterol and calcium bilirubinate in the aspirated material is confirmatory evidence of biliary tract disease

Cholangiography.—A radio-opaque solution, e.g. iodized oil, injected into the gall bladder or bile ducts, yields valuable information concerning the shape, content, and emptying of the main bile ducts. This procedure may be carried out during the operation where there is obstruction to the common duct or at a later date to ensure patency of the common duct prior to removal of the indwelling T-tube.

Pathogenesis of Gall Bladder Disease

Cholecystitis.—The experimental and clinical data on the bacteriology of the normal and inflamed gall bladder make untenable the common belief that bacterial infection is the usual cause of cholecystitis. While positive cultures have been obtained from the gall bladder wall, from the cystic lymph gland, and from the bile, there is no certain evidence that this phenomenon indicates more than secondary infection of the previously damaged viscus.

The existing evidence indicates that cholecystitis is nearly always due to chemical agents that are normally present in the body, namely, pancreatic juice and bile salts. While reflux of pancreatic juice can cause cholecystitis in man, proof is lacking that this is a common occurrence. Overconcentration of the bile salts or other constituents of the bile, which may result from partial or complete obstruction of the cystic duct, exerts a damaging effect on the mucosa of the gall bladder. The obstruction may be due to a variety of factors, e.g., anomalies

of the valves of Heister, of the cystic duct, adjacent adhesions, stone, etc. The damaged mucosa is particularly liable to invasion by pathogenic bacteria, chiefly staphylococcus, *E. coli*, streptococci, and occasionally *Salmonella typhi*.

Cholelithiasis

Chemical analysis reveals that gallstones are derived from the normal chemical constituents of the bile and are formed almost exclusively in the gall bladder. Cholesterol, calcium bilirubinate and calcium carbonate are the principal stone-forming substances. A stone may be composed almost entirely of one of these compounds, e.g., pure cholesterol stone, or a mixture of two or three.

Gallstones are likely to form when any one of the stone-forming constituents is in excess in the bile. Pigment stones composed of calcium bilirubinate are formed because of an increased excretion of bilirubin such as occurs in hemolytic jaundice. Cholesterol stones occur when there is a change in cholesterol metabolism. There is a hypercholesterolemia in pregnancy which may account for the increased incidence of cholelithiasis in multiparous women. Stones also form when gall bladder function is so altered that the solvents (bile acids) are absorbed faster than the stone-forming constituents, and this leads to precipitation and crystallization. The pH of the bile may be of some importance as an increased alkalinity tends to precipitate calcium carbonate. It seems probable, therefore, that no one mechanism can explain the occurrence of the different types of human gallstones.

DISEASES OF THE GALL BLADDER

Acute Cholecystitis

Etiology.—Acute cholecystitis is dependent in the great majority of cases upon an obstruction to the outlet of the gall bladder. In most instances this is due to a calculus impacted in the neck of the gall bladder or in the cystic duct. The obstruction

symptom is recurrent flatulent dyspepsia frequently associated with a dull, right upper quadrant pain which may radiate to the right scapula and to the right shoulder. Both the pain and dyspepsia tend to be aggravated by food of a fatty nature. Nausea and vomiting are not common

Differential Diagnosis.—Chronic gall bladder disease must be differentiated from peptic ulcer, chronic pancreatitis, disease of the upper urinary tract, recurrent appendicitis, and coronary artery disease.

Treatment.—If it can be demonstrated that chronic gall bladder disease with or



Fig 176—Cholecystograms A. Multiple radiopaque calculi. B. Laminated gallstone

Most patients with chronic disease of the gall bladder will have one or more attacks of biliary colic during the course of the disease. In its typical form, this is pathognomonic of biliary tract disease. Characteristically this pain begins suddenly in the epigastrium and right hypochondrium and frequently radiates toward the tip of the right scapula and shoulder. Nausea and vomiting usually accompany such an attack

Physical examination reveals few signs, and the diagnosis is made on a careful history with radiological confirmation by cholecystography. Tenderness over the gall bladder may be present. This tenderness may be increased if the patient is made to inspire during palpation (Murphy's sign). Fever and leukocytosis are uncommon.

without stones exists, cholecystectomy is the treatment of choice. If, however, the evidence is equivocal, the surgeon should hesitate to operate. Symptoms such as dyspepsia and flatulence are prone to persist after operation unless the pathological changes in the gall bladder are definite. The operation of cholecystostomy is rarely if ever indicated.

BILIARY FISTULA

Etiology.—Biliary fistulas are uncommon. There are three principal types

1. Spontaneous internal.
2. Spontaneous external.
3. Postoperative external

Spontaneous fistulas, either internal or external, are associated in the majority of cases with gallstones, and result from the

Treatment.—Operative treatment is indicated for all cases of acute cholecystitis, although there is wide divergence of opinion as to the optimum time for operation. If the patient is seen early, the gall bladder should be removed. This eliminates the source of infection and risk of perforation, gives a smoother convalescence, avoids the need for future operation, and can be performed without difficulty. If the patient is seen late when the symptoms are subsiding, conservative measures are preferable as they carry the least risk. Operation should not be delayed if there is increased pain, tenderness, and rigidity, and if the pulse rate, temperature, and leukocyte count do not quickly return to normal.

Cholecystostomy is indicated (1) when the patient's general condition is so serious that only the simplest and quickest operation can be considered, (2) in patients with edema or adhesions about the common duct of such a nature that a cholecystectomy is particularly difficult.

Adequate antibiotic therapy should be used in all cases. Dehydration is not uncommon, and this necessitates adequate fluid prior to operation. The glycogen and protein reserves of the liver should be replenished by the parenteral administration of the substances.

If the early treatment has been of a conservative nature, cholecystectomy should be performed at a later date when the acute process has completely subsided.

Chronic Cholecystitis and Cholelithiasis

Etiology.—In the section on pathogenesis of gall bladder disease it has been pointed out that cholecystitis is the result of chemical irritation, metabolic disturbance, or enzyme action. Infection, if present, is probably a secondary factor. Occasionally chronic cholecystitis may be a sequel to the acute form of the disease.

Pathology.—The healthy gall bladder is translucent. One of the earliest signs of pathological change is the development of opacity. In the later stages of cholecysti-

tis, the wall of the gall bladder becomes thickened and opaque. Inflammatory adhesions frequently develop between the gall bladder and adjacent viscera. The microscopic appearance may be one of fibrosis or chronic cellular inflammatory changes.

Hydrops of the gall bladder results if the cystic duct becomes obstructed in the absence of acute inflammation. The bile pigments and salts are gradually absorbed and are replaced by a watery secretion, white bile, from the mucosal surface. The gall bladder may become very distended, and may contain as much as 500 c. c. of fluid.



Fig 175—Cholecystogram. Functioning gall bladder containing radiotranslucent (cholesterol) stones.

Cholesterosis of the gall bladder is the result of a local or general change in cholesterol metabolism, characterized by the deposition of large amounts of cholesterol or cholesterol esters in the mucous membrane. The resulting small yellow specks, like strawberry seeds, have given the condition the name of "strawberry gall bladder." Stones may be present and are frequently of the pure cholesterol type. This condition does not usually give rise to symptoms.

Clinical Picture.—Cholecystitis and cholelithiasis most commonly occur in the obese middle-aged female, but are also found in males and children. The most reliable

Malignant

Carcinoma is for all practical purposes the only malignant tumor of the gall bladder. Sarcoma, melanoma and endothelioma occur but are extremely rare.

Incidence.—Primary carcinoma of the gall bladder is an uncommon tumor of the digestive tract. It occurs predominantly in females in a ratio of 4:1. This ratio parallels the incidence of cholelithiasis in the two sexes.

Etiology.—The presence of papillomas and calculi appears to be related to the development of carcinoma. While papillomas have the capacity to undergo malignant change, this transformation rarely occurs in the gall bladder. The relationship of cholelithiasis to a carcinoma of the gall bladder is interesting and probably important, as the great majority of cases show the presence of stones prior to the development of the neoplasm.

Pathology.—Carcinoma of the gall bladder is divided into two main groups: adenocarcinoma and squamous cell carcinoma. The majority of cases are varieties of adenocarcinoma, papillary, mucoid, and scirrhous. The wall of the gall bladder is thick, hard, and contracted, and at operation the condition may be mistaken for chronic cholecystitis. The tumor metastasizes early with direct spread to the liver, to adjacent organs, or along the bile passages. Distant spread may occur to lungs, bones, and other systemic organs.

Clinical Picture.—The preoperative diagnosis of carcinoma of the gall bladder is seldom made. The symptoms are usually those of long-standing biliary tract disease, although with the onset of malignancy they become more severe. Pain is a prominent feature. Occasionally the onset is insidious and the diagnosis is made only when metastases develop. Jaundice may occur from hepatic involvement or from blockage of the ducts. A palpable mass in the gall bladder region is a late sign. Acute perforation may occasionally occur.

Differential Diagnosis.—It is seldom possible to differentiate carcinoma of the gall bladder from chronic cholecystitis. Cholecystography is of little help; it merely demonstrates gall bladder dysfunction of varying degree. The advanced case must be differentiated from other neoplasms in the upper abdomen.

Treatment.—Cholecystectomy offers the only hope of cure. This procedure is frequently impossible because of the extension of the disease. The removal of the calculous gall bladder may be of value in the prevention of malignant change.

DISEASES OF THE BILE DUCTS

The clinical manifestations of biliary duct disease are the result of either

1. Obstruction.
2. Infection.
3. Functional motor disturbances.

Obstruction.—The cause of the obstruction may be

- (a) In the wall (congenital stricture, choledochus cyst, acquired stricture, inflammatory edema).
- (b) In the lumen (stone, tumor).
- (c) Outside the duct (chronic pancreatitis, carcinoma of the pancreas).

Congenital Anomalies

Congenital Cystic Dilatation of the Common Duct (Choledochus Cyst).—Choledochus cyst is a rare anomaly characterized by a localized dilatation of the common duct and probably due to a congenital weakness of the wall to which has been added an obstructive factor, such as stenosis, angulation, inflammation or achalasia.

Pathology.—The cyst is retroperitoneal and arises from the supraduodenal portion of the common duct. Its wall consists of dense fibrous tissue without a lining epithelium. The cyst varies greatly in size, may have a capacity of several liters, and contains bile-stained fluid.

erosion of a stone through the wall of the gall bladder or common duct generally into the duodenum, stomach, or colon. They may also follow penetrating peptic ulcer or carcinoma of the gall bladder, stomach, or pancreas. Most external spontaneous biliary fistulas follow an attack of acute cholecystitis. The gall bladder becomes adherent to the anterior abdominal wall, and the stoma develops by erosion through the muscle and skin. Postoperative external fistulas follow cholecystostomy, cholecystectomy, or operations on the common duct.

Clinical Picture.—In *internal fistulas* the symptoms are those of biliary tract disease. The stone may become impacted in the duodenum or terminal ileum and cause acute intestinal obstruction. Fever and jaundice may result from an associated cholangitis. Hematemesis and melena sometimes occur.

Spontaneous external fistulas occur near the umbilicus or in the right upper quadrant of the abdomen. The biliary tract should be investigated carefully in all patients who develop abscesses or sinuses in these areas. X-ray findings of air in the bile ducts or of barium in the biliary tract following a barium meal is strong evidence of biliary fistula.

Treatment.—In spontaneous fistulas and in those fistulas which follow cholecystostomy, the treatment is cholecystectomy, excision of the fistulous tract, and closure of the opening in the intestine. If intestinal obstruction is present, the offending stone should be removed. In fistulas following cholecystectomy and operations on the common duct, the tract should be excised, any biliary obstruction relieved, and a T-tube placed in the common duct.

TRAUMATIC RUPTURE

Rupture of the gall bladder or ducts may occur following penetrating wounds or trauma to the abdominal wall. Adjacent abdominal viscera are frequently involved.

The clinical picture is one of initial shock followed by a period of improvement. There

is a dull pain in the upper abdomen and within a short time the abdomen slowly distends with fluid. Jaundice due to absorption of bile from the peritoneal cavity comes on after a few days.

Treatment is usually cholecystectomy. If the tear is in the fundus it may be sutured without drainage or a cholecystostomy may be performed. If the bile ducts are injured they should be repaired over an indwelling T-tube. Any bile found in the abdominal cavity should be sucked and even washed out. Chemotherapy should be instituted.

BILE PERITONITIS

Sterile bile in the peritoneal cavity causes a chemical peritonitis which pursues a relatively benign course. However, as most cases follow operations on a diseased gall bladder, the bile is usually infected and the peritonitis while mild at first becomes progressively worse.

Etiology.—Bile peritonitis occurs following either spontaneous or traumatic rupture of the gall bladder or bile ducts or after an operative procedure.

Treatment.—Early operation is indicated. The bile in the peritoneal cavity should be aspirated, tears repaired, and a drain placed alongside the common duct so that any future leak may be aspirated. Most cases of bile peritonitis can be avoided by meticulous surgery, and drains should always be placed down to the operative site so that any bile leakage will drain to the exterior.

TUMORS OF THE GALL BLADDER

Benign

Benign tumors of the gall bladder, adenomas, lipoid polyps, and papillomas, are usually asymptomatic. Occasionally if they lie in the neck of the gall bladder in such a position as to obstruct the outlet, they may produce symptoms which mimic those of chronic cholecystitis.

Malignant

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Congenital Cystic Dilatation of the Common Duct (Choledochus Cyst).—Choledochus cyst is a rare anomaly characterized by a localized dilatation of the common duct and probably due to a congenital weakness of the wall to which has been added an obstructive factor, such as stenosis, angulation, inflammation or achalasia.

Pathology.—The cyst is retroperitoneal and arises from the supraduodenal portion of the common duct. Its wall consists of dense fibrous tissue without a lining epithelium. The cyst varies greatly in size, may have a capacity of several liters, and contains bile-stained fluid.

Clinical Picture.—Choledochus cyst occurs predominantly in the female. It rarely produces symptoms before the age of six months. The clinical features are intermittent jaundice, colicky right upper abdominal pain, and a cystic swelling in the right hypochondrium. Clay-colored stools occur.

Treatment.—The treatment of choice is anastomosis between the cyst and duodenum. If this is not feasible, a cholecystoduodenostomy may be performed.

Congenital Atresia of the Bile Ducts

The obliterative process may involve the bile channels within the liver, the hepatic ducts, the cystic duct, the common duct or the gall bladder. The involved portions may be completely absent or may be represented by cords of fibrous tissue. There is an associated widespread portal cirrhosis.

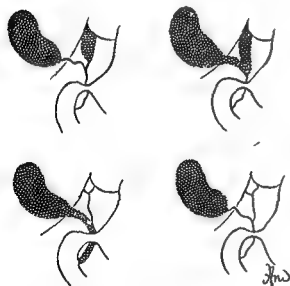


Fig. 177.—Degrees and locations of congenital atresia of the extrahepatic bile ducts

Clinical Picture.—Jaundice is present from birth, and progressively deepens for the duration of life. As the obstruction is complete, the stools are clay colored from birth. The abdomen may appear prominent due to enlargement of the liver and

spleen. Ascites is present in late cases. In the untreated case, death inevitably occurs but only after many months.

Differential Diagnosis.—The condition must be differentiated from other causes of jaundice in infancy, principally icterus neonatorum and erythroblastosis foetalis.

Treatment.—The treatment consists of an anastomosis between the proximal patent portion of the biliary system and the duodenum, although in many cases this is not possible because of the type or extent of the atresia. As in all jaundiced patients, care must be taken preoperatively to control the bleeding tendency with adequate amounts of vitamin K and transfused whole blood.

Inspissated Bile

Inspissated bile or mucus may plug the biliary ducts and give a picture that is indistinguishable from congenital atresia of the bile ducts.

Treatment.—Laparotomy with irrigation of the duct system will usually dislodge the obstructing plug and cure the condition.

Cholelithiasis

Common duct stones usually originate in a diseased gall bladder but occasionally develop within the ductal system. They may lie in the ducts or pass through the ampulla of Vater without producing symptoms. However, if they become impacted, symptoms of biliary tract obstruction occur.

Pathology.—Obstructing stones are usually single, but once obstruction occurs, secondary stones and gravel accumulate. The degree of dilatation of the ductal system depends upon the duration and completeness of the obstruction and whether it is intermittent or continuous. Mucosal ulceration occurs at the site of an impacted stone and may result in stricture or more rarely perforation. Inflammatory changes occur in the duct walls which may go on to thickening and fibrosis.

The effects on the liver depend upon the duration of the obstruction. If this is of

long standing, varying degrees of hepatocellular damage occur leading to derangement of liver function.

Clinical Picture.—The history of intermittent, colicky, right upper quadrant pain, sometimes severe; intermittent fever, and fluctuating jaundice is characteristic of biliary duct stone. The ball valve action of the stone is responsible for the intermittent character of the symptoms. Occasionally the obstruction is complete leading to a steadily deepening jaundice. The pain is more severe than that associated with acute cholecystitis, and frequently radiates through to the right scapular region and the right shoulder. It may radiate to the left hypochondrium and epigastrium. If the obstruction is complete, the pain is continuous and steady. Apart from the jaundice, there are no other physical signs of value. Roentgenological examination may or may not show the presence of the stones

If the stone does not cause obstruction, the symptoms will be simply those of the associated gall bladder disease without jaundice.

Differential Diagnosis.—The disease must be differentiated from

(a) Acute cholecystitis, acute pancreatitis, perforated peptic ulcer, renal colic, and coronary thrombosis.

(b) Other causes of obstructive jaundice such as carcinoma of the head of the pancreas and chronic pancreatitis.

(c) Intrahepatic jaundice.

Treatment.—While antispasmodics, e.g., atropine, may bring relief in the acute attack, the treatment of ductal stone is surgical. The biliary tract must be decompressed in order to relieve the jaundice and the back pressure on the liver. When the patient's condition permits, the ducts must be explored thoroughly and all stones and debris removed. In practice it is usually possible to decompress the duct and remove the stone in one operation. The ampulla of Vater should be dilated carefully to ensure free passage of bile into the duodenum.

The gall bladder, if present, should then be removed and the common duct closed over an indwelling T-tube. The preoperative preparation is of prime importance. The liver must be protected by a diet high in protein and carbohydrate. The bleeding tendency must be counteracted by parenteral vitamin K and whole blood transfusions.

Prior to the removal of the T-tube a cholangiogram must be made to ensure that no stones remain in the common duct and that the ampulla of Vater is patent.

Acquired Stricture

Etiology.—In the majority of cases, acquired stricture of the common duct is the result of injury during operations on the biliary tract. However, it is occasionally associated with ulceration of the duct due to gallstones, with cholangitis and periductal abscess, with the spread of infection from the pancreas, and with tumors.

Clinical Picture.—Jaundice is the principal clinical manifestation of ductal occlusion. The onset may be delayed for weeks or months. If the common duct has been drained, the jaundice does not appear until the T-tube is removed.

Differential Diagnosis.—Postoperative stricture must be differentiated from a retained common duct stone. This is frequently impossible without operation. However, cholangiography is of value if a T-tube remains in the common duct.

Treatment.—The treatment of acquired stricture is a most difficult surgical procedure. An effort must be made to reconstruct the ducts and re-establish continuity, and a mucosa-to-mucosa junction should be obtained whenever possible in order to prevent the occurrence of another stricture. If the gall bladder is present and functioning and the stricture is distal to the entrance of the cystic duct, an anastomosis between the gall bladder and the intestinal tract may be satisfactory.

Tumors

Tumors of the extrahepatic biliary tract are uncommon. They may arise anywhere in the ducts, but the commonest site is the ampulla of Vater.

Pathology.—Benign growths are exceedingly rare. Carcinoma is the important tumor. It is usually a diffusely infiltrating adenocarcinoma. In contrast to neoplasm of the gall bladder these tumors of the ducts are rarely associated with stone.

Clinical Picture.—The onset is insidious with jaundice as the first symptom. Dull upper abdominal pain resulting from raised intraductal pressure may accompany the icterus. The growth may ulcerate and produce gross or microscopic blood in the stool and a marked secondary anemia. The symptoms tend to be steadily progressive in contrast to those associated with stone in the common duct.

Physical findings vary with the site of the tumor. The commonest site is the ampulla of Vater when the gall bladder is commonly enlarged and palpable, unless its wall is thickened and fibrotic from a co-existent cholecystitis. The laboratory findings are those of complete obstructive jaundice.

Differential Diagnosis.—Carcinoma of the head of the pancreas, chronic pancreatitis with obstruction, and common duct stone give a similar picture.

Treatment.—In the case of a malignant tumor, a resection of the duodenum, head of the pancreas, and the involved portion of the duct should be carried out. Local resection may be done for benign growths and malignant tumors that are confined to the ampulla. In the majority of cases local spread makes these procedures impossible, and a palliative operation, such as a cholecystojejunostomy, must be done to relieve the jaundice.

Infection (Cholangitis)

Etiology.—Cholangitis is an inflammatory process which involves the walls of the bil-

iary passages and is usually associated with some obstruction to the free flow of bile. The obstruction is usually the result of stone or stricture of the common duct which predisposes to bacterial invasion. However, it may result from regurgitation of intestinal contents into the common duct.

Pathology.—The degree of cholangitis varies from a simple catarrhal inflammation to frank suppuration with resultant fibrosis and thickening. The inflammatory process may spread through the wall to involve the periductal tissues or extend upward into the tributaries of the bile ducts to involve the liver parenchyma. It may extend until all liver ducts are filled with pus.

Clinical Picture.—Acute suppurative cholangitis presents a picture of repeated episodes of chills, fever, and deepening jaundice. The liver is usually enlarged and tender. The more chronic forms do not present a definite clinical picture although they tend to flare up in the presence of obstruction, leading to further damage to the liver and ductal system.

Treatment.—The treatment is mainly prophylactic, with removal of the infected gall bladder and the relief of biliary tract obstruction. In the acute suppurative phase, the treatment is drainage of the common duct and adequate doses of antibiotics.

Biliary Dyskinesia

Biliary dyskinesia is an obstruction to the flow of bile without jaundice, due to a neuromuscular dysfunction of the sphincter of Oddi. Such a disturbance is rare in the absence of other biliary tract disease. However, in patients with biliary tract symptoms, in whom no evidence of organic disease can be demonstrated, the possibility of dyskinesia should be kept in mind.

Treatment.—The encouragement of bile flow by diet, bile salts, and antispasmodics may bring relief. Dilatation or cutting of the sphincter of Oddi has been reported to be of value.

Postcholecystectomy Syndrome

The symptoms of biliary tract disease may persist or recur following cholecystectomy or choledochostomy. Most commonly this occurs following removal of a noncalculous gall bladder, and is found in almost inverse proportion to the degree of pathological change in the gall bladder wall. Symptoms, however, can recur after cholecystectomy in properly selected cases and are usually due to residual disease. In such cases retained cystic or common duct stone, infection in the cystic duct stump, and neuromas of the periductal nerve plexus are probable etiological factors. Unfortunately only too frequently, abdominal exploration reveals no abnormality.

Treatment.—Elimination of the residual organic disease by removal of stones, amputation of the remains of the cystic duct, or periductal stripping of the nerve plexuses may give relief.

Operative Procedures

Cholecystostomy.—The indications for this procedure are limited and include:

1. Acute cholecystitis in the poor operative risk patient or when cholecystectomy presents unusual technical difficulties.

2. In certain cases of acute necrotizing pancreatitis.

3. As a first stage procedure in certain cases of jaundice due to carcinoma of the head of the pancreas or to chronic pancreatitis.

Anesthesia.—Either local, regional, or general anesthesia may be employed.

Operation.—A high paramedian or a right subcostal incision is used. Care must be taken to avoid spilling infected bile into the peritoneal cavity, and the area should be packed off before proceeding. When the gall bladder is exposed, its contents are aspirated with a wide bore needle and syringe or with a trochar and suction. The fundus of the gall bladder should be supported with two *Allis forceps* or sutures and an incision is made of sufficient length to

permit entrance of the special scoops which are used to remove the stones. Care should be taken to remove all the stones present and ensure patency of the cystic duct. A good-sized rubber tube is passed into the gall bladder and securely anchored. The opening of the gall bladder is closed about the tube, which is then brought out through the abdominal incision and connected to a receptacle.

Complications.—Approximately 40% of the patients having a cholecystostomy for gall bladder disease will have recurrent symptoms which require future operation.

Cholecystectomy.—Indications for cholecystectomy include:

1. Traumatic rupture of the gall bladder.
2. Cholecystitis and cholelithiasis.
3. Tumors of the gall bladder.
4. Internal or external biliary fistulas.

Anesthesia.—Adequate relaxation is a prerequisite to good biliary surgery. Either spinal or inhalation anesthesia is used.

Operation.—The commonly used incisions are right paramedian or right subcostal.

The gall bladder may be removed

- (a) by starting at the fundus and dissecting down to the cystic artery and cystic duct or

- (b) by preliminary ligation of the artery and duct with subsequent removal of the gall bladder. The latter procedure is usually preferable, and the former should be reserved for those cases where a large stone or adhesions tend to make clear visualization of the duct system difficult.

The fundus of the gall bladder is grasped with a pair of nontoothed artery forceps and drawn up over the edge of the liver. A second forceps is placed on the gall bladder in the region of Hartmann's pouch. When moderate traction is exerted on these forceps, the cystic duct is placed on the stretch. A small incision is made in the peritoneum over the neck of the gall bladder, and by careful dissection the whole length of the cystic duct down to its junction with the common duct is exposed. The next step

is to display the cystic artery which lies above and slightly posterior to the duct. The various anomalies must be kept in mind and great care must be taken to display and identify each structure before ligation and division.

The gall bladder is then removed from the liver bed, preserving sufficient peritoneum to cover the raw area on the liver. Soft rubber drains should always be placed in the gall bladder fossa and should not be removed for several days.

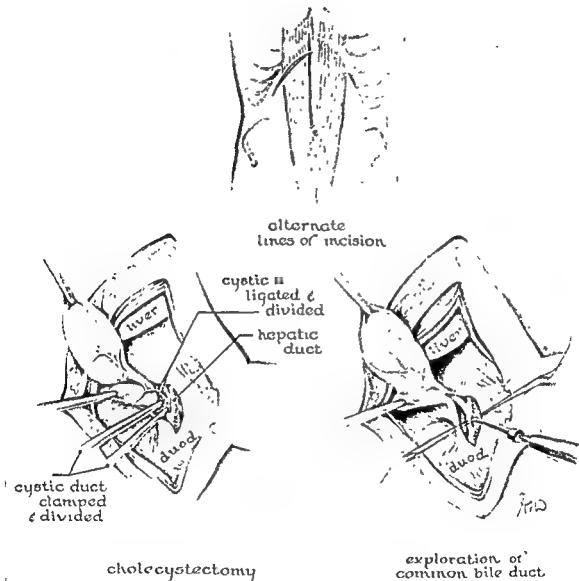


Fig 178—Technique of cholecystectomy and choledochostomy

When the dissection is complete, the artery is divided between ligatures. The cystic duct is then divided between forceps $\frac{1}{4}$ " from the common duct and doubly ligated. Care must be taken to avoid inclusion of the common duct in the ligature

Choledochostomy.—The indications for this procedure include

- 1 The presence of jaundice or a previous history of icterus.
- 2 A dilated or thickened common duct
- 3 Palpable stones in the common duct.

4. Small stones in the gall bladder with a patent cystic duct.

5 In some cases of acute pancreatitis and in those cases of chronic pancreatitis causing common duct obstruction.

Operation.—The cystic and common ducts are placed on the stretch by traction on the gall bladder. The common duct is isolated, aspirated, and the character of the bile noted. Two stay sutures of fine silk are placed on its anterior surface and a small incision is made in the duct parallel to its long axis between the two stay sutures. The duct should be irrigated to remove all sand and small stones. A probe is then passed down the duct into the duodenum to ascertain that the ampulla is patent. The ampulla should be dilated with gradu-

ated probes. The proximal portions of the ducts must be explored and irrigated to obviate leaving small stones. A T-tube is then inserted into the common duct, sutured in place, and the long limb is brought out through the incision and securely fixed to the abdominal wall. The operation is completed by cholecystectomy.

REFERENCES

- Bockus, H. L., and others: *Gastro-Enterology in three volumes*, Philadelphia, 1946, W. B. Saunders Company.
- Gatch, W. D., Battersby, J. S., and Wakim, K. G.: *The Nature and Treatment of Cholecystitis*, J. A. M. A. 132: 119-121, 1946.
- Ladd, W. E.: *Atresia of the Abdominal Stomach*, Philadelphia, 1941, W. B. Saunders Company.
- Walters, Waltman, and Snell, A. M.: *Diseases of the Gall Bladder and Bile Ducts*, Philadelphia, 1940, W. B. Saunders Company.

CHAPTER XXI

PANCREAS

H. S. DOLAN, M.D., AND J. F. HOPKIRK, M.D.

Anatomy.—The pancreas is a retroperitoneal gland which lies in the upper part of the abdomen at the level of the bodies of the first and second lumbar vertebrae. It extends from the duodenum on the right to the spleen on the left and is divided into four parts: head, neck, body, and tail. The head lies in the concavity of the duodenum, and its posterior relations are the inferior vena cava and tributaries of the portal vein. The posterior relations of the body are the aorta, superior mesenteric artery, left renal vein, left adrenal gland, and left kidney. The anterior relations of the entire gland are the stomach and the lesser peritoneal sac. The transverse colon is an inferior relation of the pancreas.

The main pancreatic duct (Wirsung) traverses the complete length of the gland from the tail to the head and usually opens into the duodenum through the ampulla of Vater. In 70% of cases it joins the common bile duct at the ampulla. Occasionally the pancreatic duct joins the common bile duct some distance from the duodenum, or the two ducts open separately into the duodenum. The accessory pancreatic duct (Santorini) enters into the duodenum 2 cm above the ampulla and rarely joins the common duct.

The blood supply of the pancreas is derived from the superior and inferior pancreaticoduodenal arteries, the splenic artery, and branches from the hepatic artery. The lymphatic distribution is rich and drains into the pancreaticoduodenal, preaortic and celiac nodes. There is also some lymphatic connection between the pancreas, gall bladder, stomach, and spleen.

The nerve supply of the pancreas is via the vagus and sympathetic systems. The

vagus is the secretory nerve of the pancreas and controls one phase of its external secretion. The sympathetic nerves (splanchnics) carry the afferent pain fibers.

Embryology.—The pancreas develops as two entodermal evaginations, dorsal and ventral, from that part of the primitive gut which goes to form the duodenum. The dorsal evagination makes up the body, tail, part of the head of the pancreas, and the accessory pancreatic duct. The remainder of the pancreatic head and the main pancreatic duct arise from the ventral evagination. The common bile duct originates from the ventral evagination which accounts for the close association of this duct with the main pancreatic duct.

Physiology.—The pancreas has two main functions:

1. The manufacture of an internal secretion (insulin), which is one of the main controlling factors in carbohydrate metabolism.
2. The manufacture of an external secretion containing a number of digestive ferments, the chief of which are lipase, trypsin, amylase, and in addition maltase, lactase, and rennin.

The insulin controls the level of the blood sugar in the body. The pancreas and the liver are the main means of control of the mobilization and utilization of glucose, and the formation of glycogen from carbohydrate sources. Secretion of insulin occurs after taking food, particularly that of a carbohydrate nature.

Removal of the pancreas results in diabetes, which is, however, less severe in its manifestations than true primary diabetes. An increase in the secretion of insulin results in hyperinsulinism which is found in cer-

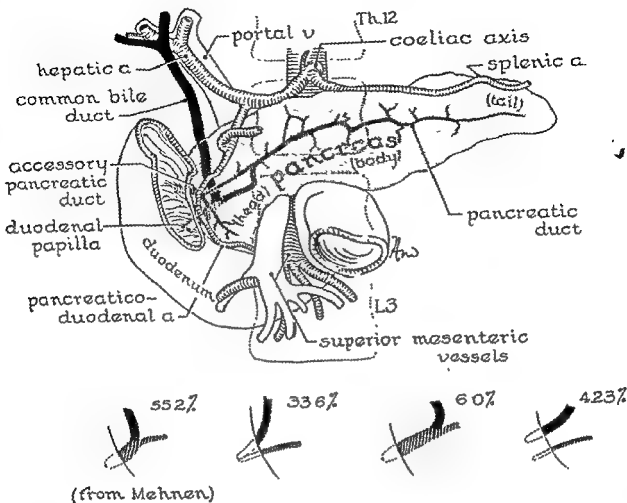


Fig 179.—The normal anatomy of the region of the pancreas. Note the intraglandular portion of the common bile duct and how easily it may be occluded by disease in the pancreatic head to cause jaundice. Note, too, the variations in the common bile and pancreatic duct junctions. From an anatomical viewpoint the common channel theory of the etiology of acute pancreatitis is tenable.

tain diseases of the pancreas, such as adenoma or hyperplasia of the islet tissue.

The external secretion is stimulated by the vagus nerve causing a secretion of a thick viscid juice which is rich in ferments, and by a hormone elaborated in the duodenum and upper jejunum, secretin, which passes via the blood stream to the pancreas where it causes a copious watery secretion of low enzyme content, containing chiefly inorganic alkaline salts.

A knowledge of the control of the internal and external secretions of the pancreas is most important when planning the therapy of patients with either acute or chronic pancreatic disease.

ABERRANT PANCREATIC TISSUE

Isolated masses of pancreatic tissue, which are completely separate from the main gland, occur in various parts of the gastrointestinal tract. Most commonly these are found in the region of the stomach, duodenum, and upper jejunum, and are located in the submucosa. Microscopically these have the characteristics of normal pancreatic tissue. They occasionally give rise to symptoms, the nature of which chiefly depends on their site. For example, when pancreatic tissue is found in the pyloric region of the stomach, the symptoms may mimic duodenal ulcer or new growth.

Occasionally the pancreas partially or completely encircles the third portion of the duodenum. If this is marked in degree, symptoms of duodenal obstruction will become evident early in life. If of minor degree, symptoms will usually not occur unless pancreatic disease develops which will secondarily cause duodenal obstruction.

LABORATORY AIDS IN DIAGNOSIS

Pancreatic Enzymes in the Blood.—The enzymes *amylase* and *lipase* are found in the blood stream of normal individuals

increased in acute pancreatitis. This, too, becomes raised early and remains at a high level for several days longer than does the *amylase*. The serum levels of both *amylase* and *lipase*, particularly the latter, may also be elevated in certain cases of malignant diseases of the pancreas.

Pancreatic Enzymes in the Urine.—The levels of urinary *amylase* and *lipase* are frequently elevated in pancreatic disease, but the estimation of the levels of these lacks much of the accuracy and specificity of the above-mentioned blood serum findings.



Fig 180—Gas and fluid levels in the small bowel in acute pancreatitis. The barium was administered in an attempt to show the enlargement of the duodenal loop

(normal serum *amylase* 80 to 200 units, Somogyi; *lipase* 85 to 205 units) The serum *amylase* level is markedly increased (500 to 1,000 units) in cases of acute pancreatitis, more usually and particularly in the early stages of the disease, and remains high for about 24 to 48 hours. After that time has elapsed, a normal serum *amylase* is of little diagnostic value in ruling out a suspected case. The serum *lipase* is also

Serum Calcium.—The estimation of the blood serum calcium level is important in cases of acute pancreatitis. The deposition of calcium, to form with fatty acids the calcium soap of fat necrosis, causes a fall in the serum calcium level. Levels below 7 mg. per ml. are indicative of a major degree of pancreatic damage and fat necrosis. Such cases usually have a very grave prognosis.

Carbohydrate Metabolism.—Disturbances in carbohydrate metabolism are very frequent in pancreatic disease. Hyperglycemia and glycosuria are often found in acute hemorrhagic or acute edematous pancreatitis. These disturbances are usually transient in nature, but they may persist following recovery, particularly in those cases in

disease, is always due to tumor or hyperplasia of the islet tissue.

Serum Bilirubin.—An elevated serum bilirubin is common during the early phase of acute pancreatitis, but clinical jaundice is unusual. However, a markedly elevated serum bilirubin indicating jaundice of the obstructive type often occurs with carci-



Fig 181.—Diffuse pancreatic lithiasis. Note the location of the pancreas at the level of L1 and L2

which marked pancreatic destruction has occurred. It is estimated that 10 to 15% of patients who recover from acute pancreatitis will later manifest impaired carbohydrate metabolism. Chronic pancreatitis, pancreatic carcinoma, and cystadenoma are also frequently associated with either glycosuria, hyperglycemia or frank diabetes. Hypoglycemia, when present in pancreatic

noma of the pancreas or chronic pancreatitis, particularly when the lesion is confined to the head of the gland. (See section on Jaundice).

Blood Count.—The white blood cell count is elevated in acute pancreatitis (15,000 to 18,000). This elevation is partly due to the accompanying hemoconcentration which is so frequently found in this condition.

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brae and is best demonstrated by an oblique, rather than an anteroposterior, view.

Barium Studies.—In acute pancreatic disease, barium meal studies may give confirmatory diagnostic evidence, if the patient is not too ill for this examination. Upward displacement of the stomach and a widening of the duodenal loop may be seen in these cases. Chronic conditions such as carcinoma of the head, cystadenoma, and pseudocyst of the pancreas will also cause a widening of the duodenal loop. In addition to this there may be evidence of extrinsic pressure which encroaches on the lumen of the stomach and duodenum.

Displacement of the colon can be demonstrated by means of a barium enema.

DEVELOPMENTAL ANOMALIES

The chief abnormalities encountered are variations of the pancreatic ductal systems and ectopic or aberrant pancreatic tissue.

INJURIES OF THE PANCREAS

Serious injuries of the pancreas are rare. When they occur, they are commonly associated with trauma to other intra-abdominal organs. They are caused by stab or gunshot wounds, underwater blasts, and severe contusions of the abdominal wall. Not infrequently minor injuries to the pancreas occur during the course of operative procedures in the upper abdomen, such as gastrectomy for duodenal or gastric ulcer, operations on the common bile duct, and splenectomy.

Occasionally pancreatic necrosis will follow a severe pancreatic injury. Pancreatic abscess, fistula, or cyst formation not infrequently develops following such injuries.

The clinical picture is that of a patient with an abdominal wound or other injury that manifests an unusually severe degree of shock. However, the diagnosis is seldom made prior to surgical exploration.

Treatment.—The treatment of serious pancreatic trauma includes the control of the associated shock and blood loss. Early operation is necessary in order to assess and repair any damage to other abdominal viscera. The management of associated pancreatic necrosis or fistula formation will be taken up under the individual sections.

ACUTE PANCREATITIS

Although some progress has been made in both the diagnosis and treatment of acute pancreatitis, the etiology of this disease remains obscure. There are two main types of acute pancreatitis:

1. Acute pancreatic necrosis or hemorrhagic pancreatitis.
2. Acute edematous pancreatitis.

It is not positively known whether or not these are separate diseases or whether they are different degrees of the same condition, but it seems probable that the latter concept is the more correct.

ACUTE PANCREATIC NECROSIS

Etiology.—The following classification indicates the numerous factors which may be the cause of acute pancreatitis (Jones):

I. Pancreatitis of infectious origin:

- (a) lymphogenous
- (b) hematogenous
- (c) extension via pancreatic ducts from duodenum or bile ducts
- (d) direct extension from infective foci or diseased adjacent viscera
- (e) activation of enzymes by bacteria in a normal gland.

II. Pancreatitis of noninfectious origin:

- (a) due to reflux into pancreatic duct of:
 - (1) Bile ("common channel" theory)—incident to:
 - (i) stone or edema of ampulla of Vater

Marked leukocytosis does not as a rule occur except in the case of suppuration following either pancreatitis or injury. If hemorrhage is severe, the red cell count and hemoglobin will fall. However, the accompanying hemoconcentration may be so marked that it masks any anemia.

Duodenal Drainage.—Examination of the aspirated duodenal contents for the presence of pancreatic enzymes, particularly following hormonal or vagal stimulation, will give

bulky, pale, and contain large amounts of undigested fat, starch, protein derivatives, and carbohydrate, due to lack of pancreatic enzyme digestion.

Urine.—The estimation of urinary enzymes, bile, and sugar, is helpful in the diagnosis of pancreatic disease and has been previously discussed.

X-ray Examination.—In acute pancreatitis a plain film of the abdomen will frequently show isolated distended loops of



Fig. 182—Carcinoma of the head of the pancreas. Note the widening of the duodenal loop, the partial occlusion of the duodenum, and the upward displacement and compression of the stomach.

information regarding the external secretion of the pancreas. Absence of these enzymes is indicative of decreased pancreatic function and occurs in such diseases as chronic pancreatitis. Cytological studies of aspirated duodenal contents may help in the diagnosis of carcinoma of the pancreas.

Examination of the Stool.—This is of value in diagnosis of chronic pancreatic insufficiency. In such cases the stools are

small bowel, usually containing fluid levels and most commonly situated in the upper midportion of the abdomen. As the disease progresses, the x-ray examination shows many large distended loops of bowel characteristic of a paralytic ileus.

Pancreatic lithiasis and calcification in the gland parenchyma can be seen in the plain film. This calcification lies at the level of the bodies of the 1st and 2nd lumbar verte-

helps to differentiate this condition from perforated peptic ulcer where rectal tenderness is usual because of the acid peptic juice which runs down into the pelvis along the paracolic gutters. There may be areas of bluish discoloration in the flank and in the periumbilical region due to extravasated blood and pancreatic ferments. This discoloration is evidence of a very severe form of the disease and as it occurs late, it is of little or no help in diagnosing either the mild varieties or the early stages of the severe form.

Diagnosis.—The signs and symptoms of the severe forms of the disease are typical. The diagnosis of the milder forms (i.e., edematous) usually requires laboratory confirmation. The serum amylase and lipase are elevated, particularly in the early stages of the disease (500 to 1,000 units).

A fall in the serum amylase with clinical improvement is usually indicative of the edematous variety. A fall in the serum amylase without clinical improvement bespeaks a very severe hemorrhagic necrosis.

The level of the blood sugar is generally elevated and that of the serum calcium depressed. The serum bilirubin is slightly elevated. There is a moderate leukocytosis and a raised hematocrit. Glycosuria may be present. A plain x-ray film of the abdomen often shows dilated loops of bowel.

Differential Diagnosis.—The disease must be differentiated from perforated peptic ulcer, early intestinal obstruction, acute appendicitis, mesenteric thrombosis, acute gastritis and coronary artery occlusion.

Treatment.—Surgical intervention is not indicated in the uncomplicated type of acute pancreatitis. The principles of treatment are fivefold: (1) to combat the pain and shock; (2) to relieve the dehydration; (3) to prevent the progression of the disease process; (4) to obviate secondary infection; (5) to correct the metabolic derangements.

1. The accompanying shock, if present, is treated by the usual measures of intravenous

infusion of whole blood and plasma. In severe cases, intra-arterial transfusions are indicated. The severe pain is relieved by the administration of morphine or Demerol. Although both these drugs can cause an increase in duodenal irritability with resultant spasm of the sphincter of Oddi, and thus contribute to the disease process by virtue of further bile reflux into the pancreas ducts, their value as analgesic agents outweighs this theoretical objection. Splanchnic block may be carried out to relieve the pain in severe cases.

2. The prolonged vomiting, together with the peritoneal exudate caused by the irritation of the disease process, and the outpouring of large quantities of pancreatic juices, produce an early and severe dehydration. This depletion must be treated by adequate amounts of glucose and water and balanced electrolyte solutions. It should be remembered that the vomiting depletes the patient of large quantities of acid gastric juice, whereas the loss of pancreatic secretion robs the patient of large amounts of alkaline ions, so that any replacement therapy must be planned to replace all types of ions. An indwelling urethral catheter is an important part of the fluid replacement program as accurate measurement of the urinary output is essential. A good renal output is the best guide for the adequacy of the fluid therapy.

3. The neurogenic phase of pancreatic secretion can be depressed by the administration of cholinergic blocking drugs. The most widely used are atropine and banthine. The hormonal phase of pancreatic secretion can be likewise controlled and minimized by measures which prevent gastric contents from entering the duodenum, with subsequent liberation of secretin, i.e., continuous gastric suction. Oral fluid in the presence of an indwelling gastric suction tube is contraindicated, as it only serves to dilute and wash out the gastric electrolytes and thus still further deplete the patient as well as increasing gastric secretion. The vagal de-

- (ii) spasm of sphincter of Oddi
- (iii) miscellaneous factors
- (2) Duodenal contents
- (b) obstruction of pancreatic ducts by:
 - (1) epithelial metaplasia
 - (2) stone in pancreatic duct or ampulla of Vater
 - (3) tumor, stricture, or edema
 - (4) duodenal diverticulum
- (c) trauma
- (d) vascular accidents: embolus, thrombosis, rupture

III A combination of two or more factors

Pancreatitis of Infectious Origin

Occasionally the disease may be due to bacteria carried by the blood or lymph stream, or in regurgitated bile. However, in most cases it would appear that any degree of infection follows only because of the secondary invasion of bacteria in the devitalized tissue of pancreatic necrosis.

Pancreatitis of Noninfectious Origin

This is the most commonly held theory. It presumes that a common channel exists between the common bile and the main pancreatic duct. Any obstruction at the ampulla of Vater, i.e., stone, edema or spasm, permits the reflux of bile from the biliary system directly into the pancreatic ducts. It is believed that the bile activates the pancreatic enzyme trypsinogen to trypsin which causes pancreatic auto-digestion with resultant thrombosis, necrosis, and release of more pancreatic enzymes. Blockage of the pancreatic ducts by hyperplasia, metaplasia, edema or stone, may be the cause in some cases.

It is noteworthy that gall bladder disease is present in the majority of cases of acute pancreatitis. Acute pancreatitis frequently follows a heavy meal or the ingestion of a large quantity of alcoholic beverages. Presumably these indiscretions bring about a copious secretion of high enzyme content,

which may, in the presence of the factors noted above, precipitate the necrosis.

Pathology.—The pancreas is enlarged, usually very soft and friable, particularly in the fulminating type although the organ may be firm in the milder forms of the disease. The surface shows necrosis, hemorrhage and areas of fat necrosis. These changes may be confined to one part of the gland, for example, the head, or may involve the entire organ. The fat necrosis appears as areas of yellowish white spots on the pancreas or on adjacent structures. Usually they are pin- or match-head in size, but are often much larger in severe cases and may be very widespread and occasionally are seen at autopsy in the pericardium or mediastinum. This fat necrosis is really not a necrotic process but is a chemical change caused by the action of the pancreatic ferment lipase which acts on neutral fat in the body, splitting it into glycerol and fatty acids. These fatty acids combine with calcium to form an insoluble calcium soap which when precipitated constitutes the characteristic lesions.

Clinical Features.—Acute pancreatitis typically occurs in middle-aged patients, most commonly in the obese and overindulgent male. There is a frequent history of a recent heavy meal or large intake of alcoholic beverages. The onset of the disease is acute, with severe abdominal pain, which is more marked in the left epigastrium and may be referred to the left subscapular region. The patient exhibits, in the severe case, all the signs of shock with rapid pulse, cold clammy skin, cyanosis, and low blood pressure. The patient shows marked physical and mental distress and early evidence of dehydration. Vomiting is severe and persistent and may be blood tinged and collapse occurs early. Abdominal tenderness is present but is not marked and is maximum in the left epigastric region. There is rigidity of the abdomen, but it is not so pronounced as in perforated peptic ulcer. Rectal examination reveals no tenderness. This fact

process, jaundice, which is caused by pressure on the intraglandular portion of the common bile duct, is the presenting symptom.

Clinical Features.—The diagnosis of chronic pancreatitis is difficult and can be proved only by operation and biopsy or at autopsy. Pain is a very common symptom, it is epigastric in position and tends to radiate to the back. This pain, however, is not pathognomonic of chronic pancreatitis and is not a reliable diagnostic feature. When jaundice occurs it is of the typical obstructive type and although pancreatic disease may be suspected to be the underlying cause, carcinoma of the pancreas cannot be excluded on clinical grounds alone. Diabetes or low sugar tolerance is not uncommon and there may be signs of deficiency in the external pancreatic secretions which is manifested by pale, bulky stools. There may be a decrease or absence of the pancreatic enzymes in aspirated duodenal contents. The levels of serum amylase and lipase may occasionally be elevated. Loss of weight is common if the disease has been established for a period of time. A plain x-ray of the abdomen may show pancreatic calcification.

A distressing feature of the disease is that it is prone to cause recurrent attacks of pain and other signs of pancreatic disease. This form is designated chronic relapsing pancreatitis.

Treatment.—In cases with severe pain or with jaundice, surgical intervention is indicated. The jaundice is relieved by decompression of the biliary system, either externally by cholecystectomy and prolonged T-tube drainage, or by an anastomosis between the biliary system and the gastrointestinal tract. The fibrotic type of chronic pancreatitis is most difficult to distinguish from carcinoma of the pancreas, even by careful examination at time of operation, and a decision to do either a conservative or radical operative procedure is never a simple one. A frozen section, if taken from

a representative area in the pancreas, will enable the surgeon to ascertain the exact nature of the condition, so that he may govern his surgical approach to the problem accordingly. Pain, which may be very severe in nature, can be relieved by bilateral splanchnicectomy in some patients. Unilateral splanchnicectomy is not recommended. Resection of the involved portion of the pancreas may be indicated for either the severe pain or the jaundice. Deficiencies in the internal and external secretion of the pancreas should be corrected by the appropriate measures, such as diet, insulin, and pancreatic ferments. Cutting or dilating the sphincter of Oddi has been recommended in the treatment of the chronic relapsing variety.

SPECIFIC CHRONIC PANCREATITIS

Specific inflammatory conditions of the pancreas, such as those due to syphilis and tuberculosis, are rare. The treatment is that of systemic syphilis and tuberculosis.

PANCREATIC LITHIASIS

The etiology of this condition is unknown, but it is probably a sequel of acute or chronic pancreatitis. Pancreatic stones are similar to salivary calculi. They are usually multiple and lie in the larger ducts. Calcification of the pancreatic parenchyma also occurs, either in conjunction with pancreatic stone or by itself.

Clinical Features.—The clinical picture is almost identical with that of chronic pancreatitis. The pain, which may be very severe and debilitating, is deep epigastric in position and radiates to the back. Nausea and vomiting are fairly common. The patient may show signs of pancreatic insufficiency with sometimes diabetes or jaundice. X-ray of the abdomen will frequently demonstrate the calculi which are usually radiopaque. Calcification of the pancreatic parenchyma is also shown by this method.

pressant agents, atropine and banthine, in addition to their direct effect on the pancreas also decrease gastric secretion and in this manner help to minimize the hormonal phase of secretion.

4. Although infection is unusual in the early stages of acute pancreatitis, it frequently intervenes later on. The devitalized pancreatic tissue forms an excellent culture medium for any bacteria in the vicinity. Infection can be controlled and even prevented by the administration of the appropriate chemotherapeutic agents.

5. A low serum calcium should be corrected by the administration of calcium salts, either lactate or levulinate. A high blood sugar should be corrected by the carefully controlled administration of crystalline insulin although overdosage of insulin should be avoided as the hypoglycemic stimulation of the vagus will increase the pancreatic secretion.

ACUTE EDEMATOUS PANCREATITIS

This form of acute pancreatitis occurs much more frequently than does the acute hemorrhagic variety. The etiology is probably identical with that of the severe form of the disease, and it is in all probability a mild form of the same condition.

Pathology.—The pancreas is edematous. There may be small areas of hemorrhage and fat necrosis, but no widespread gangrene or necrosis of the parenchyma.

Clinical Features.—The clinical picture is similar to but much less severe than that of pancreatic necrosis. It may come on following a full meal and is frequently associated with gall bladder disease. Indeed many of the patients with this disease will give a long history of previous gall bladder dyspepsia. The pain and rigidity are much less pronounced and shock is uncommon. The diagnosis is confirmed if the serum amylase is elevated. A fall in the serum amylase level co-incident with clinical improvement is good evidence of edematous pancreatitis.

Treatment.—The treatment is similar to that of acute hemorrhagic pancreatitis. Heroic supportive measures are rarely needed. Usually the relief of pain, the correction of dehydration, and the prevention of extension of the disease are all that are necessary. As gall bladder disease is frequently present, a cholecystectomy with drainage of the common duct should be carried out when the acute symptoms subside. This may be done on the assumption that the removal of the diseased gall bladder will tend to prevent subsequent attacks.

Complications of Acute Pancreatitis

Complications are more usually found following the acute hemorrhagic type and include abscess and fistula.

The treatment of pancreatic abscess is surgical drainage and appropriate chemotherapy. The treatment of pancreatic cysts and fistula is discussed later.

As a sequel to the disease, impaired glucose tolerance or frank diabetes may occur which will require dietary or insulin control. There may also be an impairment in the production of pancreatic enzymes, leading to the bulky, pale stools of pancreatic insufficiency.

Chronic Pancreatitis

It is still a matter of debate as to whether this condition is a disease in itself or a sequel of acute pancreatitis. Its etiology is unknown but it frequently occurs in association with chronic gall bladder disease which lends weight to the theory that pancreatic inflammatory disease may be due to reflux of infected bile in the pancreatic duct or perhaps infection may travel by way of the lymphatics.

Pathology.—The condition is one of fibrosis of the gland parenchyma. This fibrosis may be limited to one part of the gland or may be diffuse in its extent. The variation in the site of the fibrosis is important in that when the head is involved in the disease

ternal pancreatic secretions. In severe forms of the external variety, dehydration, due to loss of fluid and electrolytes, may occur. There is frequently a digestion of the skin of the abdominal wall in the early stages of external fistulas.

Treatment is excision of the fistula where possible or anastomosis of the tract to the gastrointestinal system.

TUMORS OF THE PANCREAS

The usual benign glandular tumors which occur elsewhere in the body are found in the pancreas; namely, adenoma, fibroma and fibroadenoma. They rarely cause symptoms and are seen as incidental findings at laparotomy or autopsy. Two forms of tumors of the pancreas are of clinical importance:

1. Islet cell tumors.
2. Adenocarcinoma.

Islet Cell Tumors

Tumors of the islet cells are frequently associated with hyperinsulinism. They do occur, however, with no demonstrable change in carbohydrate metabolism and pass unnoticed unless discovered during abdominal exploration for other conditions, or at autopsy. When hyperinsulinism is present, the symptoms are those of severe and recurrent hypoglycemia, which is manifested by sweating, flushing, pallor, dizziness, weakness, hunger, nausea, epigastric pain, syncope, and mental changes. A diagnosis of epilepsy or some circulatory abnormality is sometimes erroneously made in these cases.

Pathology.—The tumors may occur anywhere in the pancreas. They are typically reddish grey in color and are usually on the surface, but may lie deeply within the gland. Their detection at operation requires great care and patience on the part of the surgeon. These adenomas are generally benign, but the malignant variety is not uncommon. The malignant produce the same clinical picture as do the benign forms. They are

much less serious than adenocarcinoma of the pancreas as they infiltrate slowly and metastasize late.

Clinical Features.—The clinical features are those of hypoglycemia. The symptoms occur when the blood sugar falls to 50 mg. per 100 ml and are marked when it drops to 35 mg. per 100 ml. Immediate relief is obtained by oral or intravenous administration of carbohydrate. The attacks commonly occur after a period without food, i.e., a late breakfast or lack of breakfast. Increased carbohydrate metabolism caused by exercise will also bring on an attack. If the disease has been of long standing these patients are frequently very obese. This obesity is due to the fact that the patient through his own experience finds out that a high caloric intake benefits his condition. Permanent mental changes occur in severe cases.

Diagnosis.—Islet cell tumors can be diagnosed from the history, the blood sugar levels, and the clinical response to high carbohydrate feeding. The glucose tolerance curve is of limited value. If it is used, the estimation of the blood sugar should be done over at least a 7-hour period. A very low curve is indicative of hyperinsulinism but is not diagnostic. The occurrence of the attack after a period of fasting and its relief by the administration of glucose constitutes one of the best diagnostic tests.

Treatment.—Surgical removal of the hyperfunctioning tumor is essential. Occasionally a diffuse hyperplasia of the islet cells causes hyperinsulinism. In this case the treatment is radical subtotal resection of the pancreas. The results of excision of the pancreatic adenoma are good if irreversible mental changes have not occurred.

Carcinoma of the Pancreas

The only malignant tumor of clinical importance other than the islet cell carcinoma is adenocarcinoma. It has been more often encountered in recent years because of more

Treatment.—The treatment is surgical and consists of removal of the stone in cases where this is possible, or pancreatic resection in those cases with diffuse calcification. As in ordinary chronic pancreatitis, bilateral splanchicectomy will give relief of pain in some cases. Attention must be paid to insufficiencies of the pancreatic secretions.

PANCREATIC CYSTS

Pancreatic cysts are not common; the most frequently occurring variety is pseudocyst of the pancreas, which develops as a complication of acute pancreatitis or pancreatic injury. The following classification indicates the types which have been reported. (Mahorner and Mattson.)

- I. Cysts resulting from defective development.
 - (a) cysts in infants,
 - (b) cysts associated with polycystic disease of the kidney,
 - (c) dermoid cysts,
 - (d) inclusion cysts
- II Cysts resulting from trauma
- III Retention cysts.
- IV Neoplastic cysts,
 - (a) cystadenoma,
 - (b) cystadenocarcinoma,
 - (c) teratomatous cysts
- V Cysts resulting from parasites.

Pathology.—Pancreatic cysts may occur in any part of the gland.

The three most common and most important cysts of the pancreas are:

- 1 Cystadenoma of the pancreas
- 2 Fibrocystic disease of the pancreas
3. Pseudocyst of the pancreas.

Clinical Features.—The clinical features of pancreatic cysts in adults are the same regardless of the exact nature of the cyst. There is frequently a history of acute pancreatitis or trauma to the abdominal wall or of abdominal operations. The majority of cysts, however, are not noticed until they

form a readily palpable mass. In the later stages there is vague pain, loss of weight, nausea and vomiting. Rarely jaundice occurs due to extrinsic pressure on the biliary system. Impaired glucose tolerance, diabetes, and insufficiency of external secretion may occur.

Cystadenoma of the pancreas is a large polycystic tumor with papillary projections from its columnar cell lining. It rarely gives rise to symptoms until it becomes large enough to cause pressure on the surrounding structures. Malignant forms occur. The treatment is surgical removal.

Fibrocystic disease of the pancreas occurs in infants and children. It is characterized by widespread fibrocystic changes in the pancreas, which are usually associated with similar changes in other organs, notably the lungs and kidneys. There is complete loss of external pancreatic secretion due to malformation of the ducts which gives rise to the condition known as meconium ileus.

Treatment.—Laparotomy is usually required to relieve the intestinal obstruction. Pancreatic enzymes should also be given. The prognosis is unfavorable because of the associated fibrocystic disease in other organs.

Pseudocysts or false cysts are effusions into the lesser peritoneal sac which become sealed off by adhesions. Occasionally these cysts contain blood because of small hemorrhages from the vessels either in the pancreas or the cyst wall. They occur following acute pancreatitis or trauma.

Treatment is excision of the cyst, gastro-intestinal pancreatic anastomosis, or marsupialization of the cyst wall.

PANCREATIC FISTULA

These fistulas may be internal or external. The diagnosis of the external variety is obvious and is the only one of clinical importance. There is a history of acute pancreatitis or surgical operation. There may be insufficiency of either the internal or ex-

- Gray, H. K.: Carcinoma of the Pancreas, Arch. Surg. 57: 763-773, 1918.
- Jones, R., Jr.: Etiology and Pathogenesis of Acute Hemorrhagic Pancreatitis, Am. J. M. Sc. 205: 277-301, 1943.
- Kruger, R., and Dockerty, M. B.: Tumors of the Islets of Langerhans, Surg., Gynec. & Obst. 85: 495-511, 1947.
- Mahorner, H. R., and Mattson, H.: Etiology and Pathology of Cysts of the Pancreas, Arch. Surg. 22: 1018-1033, 1931.
- McDonough, F. E., and Heffernan, E. W.: Chronic Lapsing Pancreatitis, S. Clin North America 28: 731-740, 1918.
- Meyer, K. A., et al.: Pseudocysts of the Pancreas: Report of 31 Cases, Surg., Gynec. & Obst. 88: 219-229, 1919.
- Ray, B. S., and Console, A. D.: The Relief of Pain in Chronic (Calcareous) Pancreatitis By Sympathectomy, Surg., Gynec. & Obst. 89: 1-8, 1919.
- Rich, A. R., and Duff, G. L.: The Etiology and Pathogenesis of Acute Pancreatitis, Bull. Johns Hopkins Hosp 58: 212, 1936.
- Whipple, A. O.: Radical Surgery for Certain Cases of Pancreatic Fibrosis Associated With Calcareous Deposits, Ann. Surg. 124: 991-1008, 1916.

accurate diagnosis and the growing awareness of this condition

Pathology—The tumor is an adenocarcinoma, usually scirrhous in type which arises from the epithelium of the duct system. Occasionally it is medullary in type. The neoplasm develops in either the head or tail or it may involve the entire gland.

Clinical Features.—The picture varies with the position of the tumor. Jaundice occurs early in carcinoma of the head as it causes pressure on the intraglandular segment of the common bile duct. The usual symptoms of carcinoma, namely, loss of weight, loss of strength, and loss of appetite, are present. Abdominal pain, usually epigastric, is frequent and is of boring character and generally radiates to the back. Carcinoma of the tail or body of the pancreas does not produce jaundice unless it is very advanced. Pain comes on early and is typically the first sign. The tumor is rarely palpable. Ascites, when it occurs, is a late complication and is caused by portal vein obstruction due to lymph node involvement. The gall bladder is frequently palpable in carcinoma of the head of the pancreas. In addition to these signs and symptoms, there may be decreased sugar tolerance or diabetes, glycosuria, and diminution of external pancreatic secretion as well as a slight elevation in the serum lipase.

Diagnosis—The diagnosis is frequently difficult. It is made from the history, the presence of jaundice, and the employment of x-ray and other laboratory procedures such as examination of duodenal contents for enzymes and tumor cells, estimation of serum lipase levels and examination of the stools for presence of blood and evidence of pancreatic insufficiency. There may be an impaired carbohydrate metabolism. The typical x-ray finding in carcinoma of the head of the pancreas is a widening of the duodenal loop due to pressure when visualized by means of barium studies.

Treatment.—To date there has been no satisfactory form of treatment. Many tech-

niques for resection of the involved portion of the gland with reconstitution of the gastrointestinal, biliary, and pancreatic flow have been devised. Such procedures are of value in that they relieve jaundice and thereby make the patient more comfortable. However, the operation is one with a significant mortality, and because of the fact that carcinoma of the pancreas infiltrates rapidly and metastasizes early, it is usually impossible to resect the entire area of malignancy. It is possible that a minute tumor could be cured by radical resection, but unfortunately by the time carcinoma of the pancreas is clinically obvious, it is no longer minute and is no longer amenable to curative surgery. Palliative treatment for severe jaundice with all its accompanying discomforts is helpful. This consists of drainage of the biliary system either externally or by biliary-intestinal anastomosis. The pain may be relieved by analgesics. It is not recommended that splanchicectomy be carried out for the relief of pain in these cases.

The duration of life is short once the diagnosis has been made (six to twelve months).

REFERENCES

- Cattell, R. B., and Pyrtek, L. J.: Appraisal of Pancreatoduodenal Resection: a Follow-Up Study of 61 Cases, *Ann Surg* 129: 840-849, 1949.
- Cole, W. H., and Reynolds, J. T.: Resection of the Duodenum and Head of the Pancreas for Primary Carcinoma of the Head of the Pancreas and Ampulla of Vater, *Surgery* 18: 133-143, 1945.
- DeTakats, Goza, and Walter, L. E.: The Treatment of Pancreatic Pain by Splanchnic Nerve Section, *Surg, Gynec & Obst* 85: 1-8, 1947.
- Doubilet, Henry, and Mulholland, J. H.: Recurrent Acute Pancreatitis: Observations on Etiology and Surgical Treatment, *Ann Surg* 128: 609-638, 1948.
- Doubilet, Henry, and Mulholland, J. H.: Surgical Treatment of Pancreatitis, *Surg Clin North America* 29: 339-359, April, 1949.
- Dozzi, Daniel L.: Acute Pancreatic Necrosis and Hemorrhage, *Pancreatic Hemor-*
- Elatson, E. L., and Welty, R. F.: Pancreatic Calculi, *Ann Surg* 127: 150-157, 1948.

ANATOMY AND PHYSIOLOGY

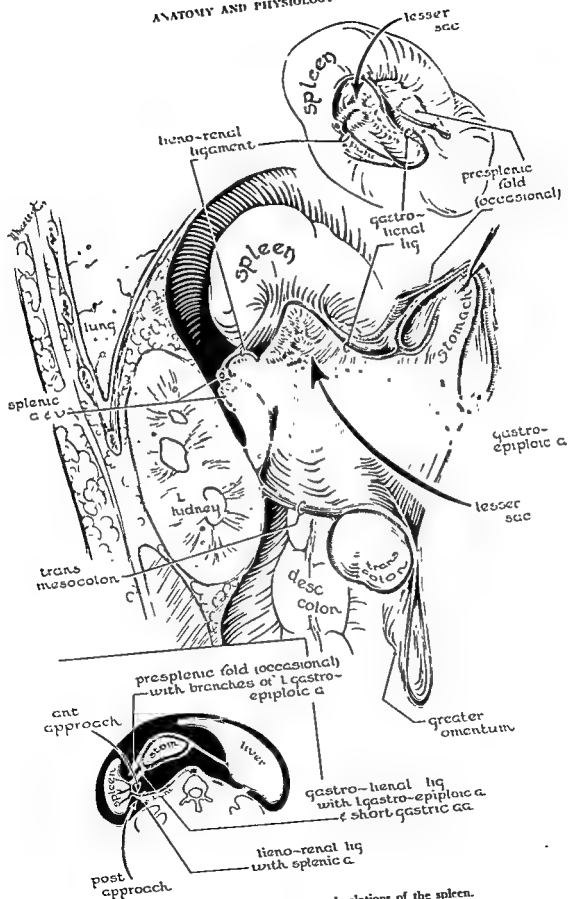


Fig. 183 — Peritoneal relations of the spleen.

CHAPTER XXII

SURGERY OF THE SPLEEN

DAVID W. MACKENZIE, JR., M.D., AND LOUIS LOWENSTEIN, M.D.

Introduction

With few exceptions splenectomy is the only surgical procedure involved in diseases of the spleen

It is stated that splenectomy was performed on Marathon contestants of the last century B.C. in order to increase their stamina. It is not improbable that a high percentage of individuals so treated were handicapped by massive malarial spleens and, therefore, benefited materially, in a purely mechanical sense, from the removal of such awkward abdominal tumors. In what manner and how frequently it was then possible to compass the technical hazards of operation remains to this day a matter for conjecture.

There are few authenticated records of splenectomy during the Middle Ages and Renaissance period. In 1856, Adelman of Berlin reported 15 cases, but failed to mention the underlying pathological conditions. In 1880, Pean is said to have removed the first splenic cyst. By 1900, Bessel-Hagen had operated upon 37 ruptured spleens. Since the turn of the century, splenectomy has followed the general trend of technical improvement and has shared in the security of improved supportive treatment. It is performed with ever-increasing frequency and decreasing mortality. To the traditional indications for this operation—rupture, ectopy and primary tumor—the expanding science of hematology has added various blood dyscrasias, several within the past five years. However, those of broadest experience will admit that removal of the spleen may be at times impossible and that the consequences of ill-advised surgical interference may be rapidly fatal.

Galen spoke prophetically when he described the spleen as *an organ full of mystery* and Stukeley's opinion (1723), that *what formerly was the seat of joy has become a topic of grief to the moderns*, is doubtless shared by many surgeons of our time.

Applied Anatomy

The spleen is several times larger in life than in death. Its normal autopsy weight is about 100 grams. It is almost completely invested by peritoneum, of which various folds constitute suspensory ligaments, gastrosplenic, phrenicosplenic, lienorenal and phrenicocolic. The length of these peritoneal reflections determines the mobility of the spleen, which is subject to marked variation. The phrenicosplenic and phrenicocolic ligaments and the lateral fold of the lienorenal ligament are normally avascular. The gastrosplenic ligament contains the short gastric and left gastroepiploic branches of the splenic artery and the lienorenal ligament enfolds the true splenic pedicle. In congestive splenomegaly, all the ligaments are highly vascular and the spleen is often plastered to the diaphragm by tough adhesions rich in collateral vessels. The peritoneal relationships of the true pedicle make easier the posterior approach to the vessels. From behind there is only one intervening layer of peritoneum; from the front, at least three, sometimes five.

The spleen lies in the cupola of the left diaphragm. The ninth, tenth and eleventh ribs overlie its convex surface. Because of its situation, friability and vascular turgidity, it is easily ruptured by direct or indirect violence. Anteromedially, the spleen is closely apposed to the stomach; postero-

latter open. Direct observation of the microscopic circulation of certain mammalian spleens has led us to favor the hypothesis of a morphologically open system which, from the standpoint of function, may be closed rhythmically or in response to those stimuli which cause arterial constriction.*

*Work being presently (March, 1952) carried on by A. O. Whipple and A. K. Parpart in the Princeton Biological Laboratory appears to confirm the impression we gained by transillumination in 1941 and is likely soon to provide televised proof of an open circulation.

Applied Physiology

It is generally accepted that the spleen participates in the production, destruction, storage, and filtration of blood. While many of the details of these mechanisms are still obscure, many more have been clarified by recent investigation.

Reservoir Function.—Since the classical experiments of Barcroft and his colleagues,

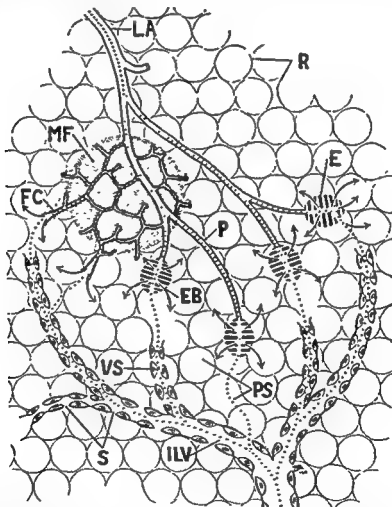


Fig. 185.—Diagram of the intralobular circulation in certain mammalian spleens (e.g., that of the cat), as suggested, in part, by McNee's representation of a splenic lobule. Arrows radiating from ellipsoids and follicle capillaries suggest a few of the innumerable pathways by which blood may traverse the pulp interstices in relaxed or distended spleens, and emphasize the morphologically "open" character of the circulation. Dotted lines indicate the short-cuts taken by most of the blood, when the circulation is functionally "closed" by splenic contraction. LA, lobular artery; MF, Malpighian follicle; P, penicillus; FC, follicle capillary; E, ellipsoid; EB, lateral channel in wall of ellipsoid; R, red pulp reticulum; PS, pulp spaces; VS, venous sinus; ILV, intralobular vein, S, stigmata in walls of venous sinus and intralobular vein. This diagram, constructed by one of us (D. W. M.), was originally published in the *American Journal of Anatomy*, Vol. 68, page 445.

medially, to the left kidney, the tail of the pancreas and the colon. Any of these structures is liable to injury during splenectomy.

The splenic artery lies above the splenic vein, behind the omental bursa and parallel with the upper border of the pancreas. For purposes of ligation, it may be approached through either the gastrohepatic or the gastrotocolic omentum.

erythrocytes, lymphocytes, monocytes, clasmatoocytes and granulocytes. Anastomosing, cylindrical strands of lymphoid tissue appear in sections as Malpighian corpuscles. Each lymphoid cylinder surrounds a lobular branch of the splenic artery. The sharp demarcation of splenic infarcts confirms the infrequency of vascular anastomoses. Though it has been described as a giant

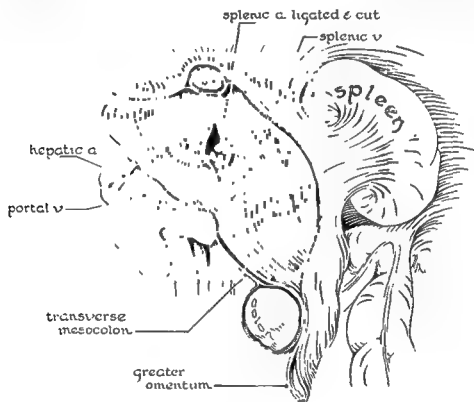


Fig 184—Ligation of splenic artery

Microscopic Features.—Besides its peritoneal covering, the spleen possesses a fibro-elastic coat from which trabeculae, penetrating in all directions, form the framework which supports the splenic pulp in its interspaces. The capsule and trabeculae of the human spleen are highly elastic but contain very little nonstriated muscle. Contractility is therefore passive rather than active, in contrast to what is found in laboratory animals. The pulp is a spongy, fibro-cellular feltwork of reticulum, of which the meshes contain varying proportions of

hemolymphnode, the spleen contains no lymphatic vessels

The microscopic *vascular anatomy* of the spleen remains a problem. The point of controversy is whether or not the reticulo-endothelial components of the pulp are directly exposed to the circulating blood; whether, in other words, the arterial and venous capillaries of the spleen consist of intact endothelial tubes in unbroken continuity, or whether a zone of naked pulp intervenes between them. The former type of circulation has been called *closed*, and the

selectively trapped in, and destroyed by, the spleen. Though the function of iron storage has been attributed to the spleen, it is not clear that this mineral is retained in excess of the quantity derived from hemolyzed or phagocytosed erythrocytes. In certain diseases associated with excessive blood destruction, abnormal red cells are trapped in the spleen, filtered from the general circulation, and destroyed by increased phagocytic activity. Leukocytes and blood platelets also are phagocytosed in some cases of hypersplenism.

Antibody Production.—Resistance to some infectious diseases is thought to be decreased after splenectomy. Recent evidence indicates that antibodies may be elaborated by and concentrated in the spleen; that, for example, the spleen is the major site of antibody production in some of the acquired hemolytic anemias, thrombocytopenic purpuras, splenic neutropenias and hematompenias. In this group of immunocytopenias the reservoir function, filtration mechanism, hematocrit, stagnating and phagocytic capacities of the spleen enhance the damaging effects of the antibodies. Whether the production of certain antibodies is peculiar to the spleen or whether, in some cases, the spleen merely contributes its share as part of the reticulo-endothelial lymphocytic and plasmacytic systems remains to be clarified.

In conclusion, the spleen is not essential to life, nor is its removal necessarily followed by any serious or permanent disturbance. Such changes as have been described are inconstant; in fact, available evidence suggests that the spleen is more important pathologically than physiologically.

INDICATIONS FOR SPLENECTOMY

If more were known about the functions of the spleen, the indications for its removal could be stated with greater certainty. In cases of trauma, congenital ectopy, and mechanical exigency, the responsibility is purely surgical. Otherwise, the problem is to interpret the mechanism of imbalance be-

tween the hematopoietic functions of the bone marrow and the inhibitory and destructive activities of the spleen. Such interpretation, as has been amply demonstrated, demands the cooperative efforts of a team of hematologists, internists, and surgeons. Adequate preoperative investigation will eliminate most of the pitfalls and disasters of splenectomy. Prolonged follow-up, including frequent hematological studies, will provide the only accurate assay of its results.

Generally speaking, the indications for splenectomy may be tabulated as follows:

Group I.—As a rule, the following conditions are absolute indications:

- (a) Rupture of the spleen.
- (b) Wandering or ectopic spleen.
- (c) Hereditary spherocytic anemia
- (d) Thrombocytopenic purpura hemorrhagica.
- (e) Primary splenic hematopenia.
- (f) Congestive splenomegaly caused by obstruction of the splenic vein.
- (g) Primary neoplasms of the spleen.
- (h) Aneurism of the splenic artery.

Group II.—Splenectomy may be required for:

- (a) Cystic disease of the spleen.
- (b) Abscess of the spleen.
- (c) Granulomatous infections or parasitic infestations localized to the spleen.
- (d) Gaucher's disease.
- (e) Acquired hemolytic anemia.
- (f) Hypersplenism secondary to a variety of causes
- (g) Splenomegaly of indeterminable etiology.

Group III.—Splenectomy may expedite major surgical procedures involving:

- (a) Esophagus, stomach and pancreas, especially in block resections for malignant disease.
- (b) Tributaries of the portal bed, in shunting operations for the relief of portal hypertension.

it has been thought that the spleen serves as a reservoir of red cells, which are released into the general circulation in response either to oxygen lack, caused by violent exertion, hemorrhage, asphyxia, diminished atmospheric pressure or carbon monoxide poisoning, or to any adrenergic stimulus that elicits contraction of the spleen. Anatomically, especially in those animals whose trabeculae contain an abundance of smooth muscle, the spleen is admirably adapted to mobilize its content of blood. In the normal human spleen there is doubt concerning the practical importance of this function, for it has been shown that, following exercise or injection of adrenalin, hematocrit values increase in splenectomized individuals comparably to those with intact spleens; that in both there is an associated decrease in plasma volume. In certain diseases, however, usually associated with splenomegaly, enhancement of reservoir function may have an important bearing upon the pathologic physiology of the disease; for example, in spherocytic anemia, hereditary or acquired sickle cell anemia, thalassemia major, and splenic hematopenia, the reservoir function of the spleen has been subdivided in a manner comparable to that of pulmonary function, namely, into complemental, tidal, reserve and residual blood components.

Hematopoiesis—Red cells are produced in the spleen during the first two trimesters of fetal life. Following birth, erythropoiesis in the spleen normally ceases. In certain diseases, associated with impaired blood production in the bone marrow, erythropoiesis and myelopoiesis may take place. Throughout life, lymphocytes are formed in the Malpighian corpuscles, and reticuloendothelial elements give rise to monocytes. Occasionally, in the presence of marrow failure, megakaryocytes are found in the spleen.

Remote Control of Hematopoiesis.—There is evidence that the normal spleen exerts a braking or inhibitory influence upon hematopoiesis in the bone marrow and also

controls to some extent the maturation and possibly the delivery of red cells to the circulating blood.

To date, most of the evidence for this remote effect of the spleen has been based upon studies of bone marrow and blood before and after splenectomy. In normal persons, removal of the spleen is usually followed by anemia, leukocytosis, thrombocytosis, reticulocytosis, the appearance of nucleated red cells, Howell-Jolly bodies and Cabot rings, basophilia, polychromatophilia, target cells, and increased resistance of erythrocytes to hypotonicity. Anemia is usually mild and transient, but Howell-Jolly bodies have been observed in the red cells many years after splenectomy. The life span of transfused normal red cells is not affected by removal of the spleen. Leukocytosis may persist for years. At first, it is chiefly a neutrophilia. Subsequently the proportion of lymphocytes and monocytes increases. Eosinophilia and basophilia are occasional findings.

Recently, hyposplenism has been observed in nontropical sprue. In such cases, target cells and Howell-Jolly bodies were found, pointing an analogy to the effects of splenectomy in the normal individual, and suggesting the possibility that functional *hyposplenism* as well as *hypersplenism* may occur.

Filtration of Blood.—The capacity of splenic sinusoids and pulp spaces to separate blood cells from blood fluid is readily observed in transilluminated spleens. The term *hematocrit function* aptly describes this activity. The reticulo-endothelial components of the pulp, by adsorption and subsequent phagocytosis, immediately remove foreign particulate matter from the fluid which bathes them. This process, which is practically instantaneous, is strong evidence against the presence of a limiting membrane between the circulating blood and splenic pulp.

Blood Destruction.—There is abundant evidence that old or abnormal red cells are

Clinical Features.—The wandering spleen is diagnosed by its shape and by the fact that palpation will sometimes return it to its normal bed. It may be mistaken for a pedunculated pelvic tumor, a mobile kidney, or any obscure intra-abdominal mass. The symptoms are those of local pressure effects: a dull ache, a dragging sensation, and dyspepsia. When the pedicle becomes twisted sufficiently to interfere with the blood supply, pain is severe and associated with prostration, vomiting and distention, as in the case of twisted ovarian cyst.

Treatment.—Complicated or not, the wandering spleen should be removed. Palliative procedures, such as splenopexy, are no longer approved.

HEREDITARY SPHEROCYTIC ANEMIA

Synonyms.—Chronic acholuric jaundice, congenital or familial hemolytic icterus, and hereditary globe-cell anemia.

Definition.—A form of hereditary hemolytic anemia transmitted as a simple Mendelian dominant, characterized by jaundice, anemia, splenomegaly, spherocytosis, and increased fragility of red cells to hypotonicity. Hemoclastic crises, which are acute exacerbations of the hemolytic process, frequently occur.

Clinical Features.—Symptoms usually develop early in life. The blood of other members of the family may show traits of the disease. Anemia and icterus may be mild or severe; the patient is usually more yellow than sick. The onset may be initiated by an acute hemolytic crisis with pain in the back, abdomen, and limbs, severe malaise, chills and fever, prostration, and occasionally circulatory collapse, oliguria, and even anuria. If the anemia is severe, dyspnea, palpitation, and other circulatory symptoms are prominent. Hemoglobinemia and hemoglobinuria are very rare. The urine may contain albumin and casts.

The indirect plasma bilirubin is elevated and urobilinogen excretion is increased in the stools and urine. The anemia is only severe during hemoclastic crises. Temporary hypoplasia or aplasia of bone marrow with rapid increase of anemia and decrease of reticulocyte, leukocyte and platelet counts in the blood without significant increase of jaundice may occur early in an acute hemolytic crisis, especially if the crisis is precipitated by infection. There is a variable reticulocytosis, highest after severe hemolysis. The characteristic red cell is the *spherocyte*, a densely staining cell with decreased diameter and increased thickness as compared with normal erythrocytes. These spherocytes show increased fragility to hypotonic solutions and mechanical tests. The Coomb's test for coating antibodies is usually, but not invariably, negative (see Acquired Hemolytic Anemia). Although usually normal, white cells and platelets may be slightly decreased in number. The bone marrow shows normoblastic hyperplasia. Infection, trauma, fatigue, emotional crises, exposure to cold, and occasionally pregnancy, may precipitate an acute hemoclastic crisis. Cholelithiasis occurs in approximately two-thirds of cases. Radiological changes in the bones may be found when anemia and jaundice are of long duration. The spleen is large and firm. Hepatomegaly is frequently observed and the function of the liver may be impaired. Congenital abnormalities occur with unusual frequency.

Diagnosis.—The various causes of jaundice, fever, anemia, abdominal pain, splenomegaly, and cardiovascular disease must be considered. Cholelithiasis may obscure the primary diagnosis or may precipitate and be hidden by an acute hemolytic crisis. The anemia is usually microcytic and sometimes the fragility of the red cells is normal. A false positive Wassermann reaction may be obtained.

Complications.—Cholelithiasis and acute hemolytic crises are the principal complications. Preferably, splenectomy should pre-

CONTRAINDICATIONS OF SPLENECTOMY

Splenectomy is contraindicated in the following conditions:

- (1) Leukemia, lymphatic or myeloid, unless marked hypersplenism is present.
- (2) Polycythemia vera.
- (3) Splenic metaplasia, associated with:
 - (a) Secondary carcinoma of the bone marrow.
 - (b) Osteomyelofibrosis
 - (c) Marble bone disease.
 - (d) Refractory anemias resulting from various forms of intoxication (e.g., benzol or radium dial poisoning, or the effects of certain radioactive isotopes).

RUPTURE OF THE SPLEEN

Rupture of the spleen may be caused by direct or indirect violence, or it may occur spontaneously. Its effects are either immediate or delayed. Diseased spleens, especially those of malaria, infectious mononucleosis and acute generalized infections, are far more liable to traumatic or spontaneous rupture than is the normal organ. It must be remembered, however, that the healthy spleen is more readily lacerated than any other abdominal viscus and that the injury may have been sufficiently trivial to be overlooked by the patient.

Clinical Features.—The signs and symptoms of ruptured spleen are those of internal hemorrhage together with peritoneal irritation. Shock may be immediate. There is abdominal pain, usually more marked in the left hypochondrium and flank. There is often reference of pain to the left shoulder. Muscular resistance is variably increased. Dullness to percussion may be present, shifting on the right but constant on the left side. The picture is frequently complicated by concomitant injury to other organs.

The initial phase may progress rapidly to a fatal termination. Fifty per cent of untreated patients die within an hour of in-

jury. Bleeding may respond promptly to rest, sedation and one or more blood transfusions, or it may continue slowly and persistently. Delayed hemorrhage is a dangerous complication of untreated rupture of the spleen. It occurs suddenly and without warning, hours, days, or even months following the injury.

Treatment.—If massive intra-abdominal hemorrhage is suspected, laparotomy must be undertaken as soon as maximum resuscitation has been achieved. Rupture of the spleen will be encountered in over 30 per cent of all severe abdominal injuries. An upper left paramedian incision is preferred. The ruptured spleen, which is usually abnormally mobile, is delivered into the wound and after its pedicle is secured and tied off, the organ is removed with the least possible delay. A search for other injuries is then carried out; the peritoneal cavity is cleaned, and the abdomen closed. The operative mortality in cases of ruptured spleen is at least 10%; of delayed rupture, 20%. Acute splenic rupture untreated causes death in over 75% of cases.

ECTOPIC SPLEEN

Ectopia of the spleen is a rare condition. It may be congenital, due to anomaly or absence of the supporting peritoneal ligaments, or acquired as a result of trauma which ruptures the phrenicocolic band, or because of splenic enlargement which stretches and abnormally mobilizes the peritoneal folds. A wandering spleen may be found in any part of the abdominal cavity, or it may be contained within a diaphragmatic or external hernial sac.

The important complication of this condition is twisting of the pedicle, which leads to hemorrhagic engorgement, necrosis, rupture or atrophy. The wandering spleen, which, because of chronic perisplenitis incidental to vascular engorgement, has become fixed in some abnormal position, is said to be *dislocated*.

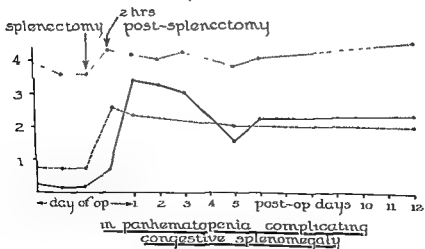
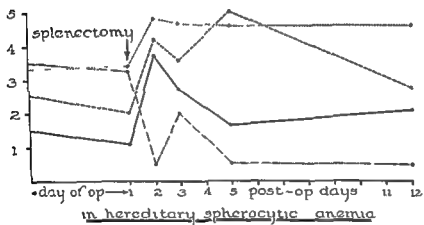
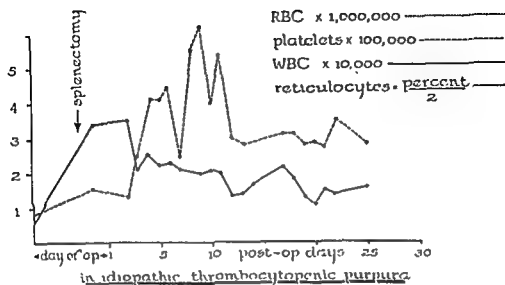
Hematological Effects of Splenectomy

Fig 186 — Hematological effects of splenectomy in

- idiopathic thrombocytopenic purpura,
- hereditary spherocytic anemia,
- splenic panhematopenia in congestive splenomegaly.

cede biliary surgery. Infrequently, anuria develops during a hemolytic crisis. Leg ulcers may occur and heal only after splenectomy.

Pathology.—The spleen usually weighs between 800 and 1,500 grams, although we have recently removed one of 3,500 grams. Perisplenic adhesions are common. The cut surface of the spleen is purplish. Malpighian bodies are small and widely separated. Hemosiderosis occurs and, in supravital preparations, increased phagocytosis may be observed. The bone marrow shows a normoblastic hyperplasia. Most authorities now agree that the pathogenesis of this disease is an hereditary defect in the bone marrow, characterized by spherocytosis of adult erythrocytes. Such abnormal red cells are selectively destroyed by the spleen. The mechanism of destruction is unknown. A possible explanation is that the unique structure of the splenic pulp is particularly adapted to the mechanical trapping of spherocytic red cells, in the same manner in which it disposes of ageing normal erythrocytes, which tend to become spheroidal.* This specific splenic hemolysis, in which the spleen apparently functions independently of the rest of the reticulo-endothelial system, occurs consistently only in the hereditary variety of spherocytic anemia. Red cells, from patients with hereditary spherocytic anemia, have a shorter than normal life span, both in their own circulation and when transfused into normal recipients. Their life span is increased both in splenectomized normal recipients and after splenectomy in patients with this disease. Normal red cells transfused into a patient with hereditary spherocytic anemia have a normal life span.

Treatment.—Splenectomy is almost invariably followed by the disappearance of anemia and jaundice. Contraction of the spleen prior to its removal may increase the

red cell count by half a million or more per cubic millimeter. During the next few days, temporary decrease of red cells and hemoglobin is followed by gradual return to normal. Leukocytosis occurs, but is usually transient. A marked thrombocytosis probably accounts for the high incidence of post-operative thrombotic phenomena. If splenectomy does not achieve the desired result, retention of accessory spleens must be suspected. In some instances, subsequent removal of accessory spleens has resulted in permanent clinical cure. As a rule, spherocytosis and increased fragility are unaffected by splenectomy.

In this disease, preoperative transfusion of whole blood was formerly held to carry with it grave risk of serious reactions. It is our impression, however, that recent advances in blood grouping and transfusion technique have eliminated much of this hazard. Few cases of hereditary spherocytic anemia require preoperative transfusions.

It has been stated that splenectomy is contraindicated during an acute hemoclastic crisis. Obviously, the operative risk is increased during an exacerbation of this disease, but so is that of expectant treatment. Current opinion generally supports the view that the results of surgical intervention are considerably better than those of nonsurgical supportive measures.

THROMBOCYTOPENIC PURPURA HEMORRHAGICA

Synonyms.—Idiopathic thrombocytopenic purpura, Werlhof's disease, thrombocytolytic purpura, hemogenic syndrome.

Definition.—A condition of unknown etiology, most commonly occurring in children and in young adult females, characterized by thrombocytopenia and spontaneous bleeding. There is no evidence of other primary disease of the skin or hematopoietic system. Spontaneous remissions and exacerbations are characteristic. At times, there is a familial tendency. The disease is relatively uncommon in the Negro.

*Whipple and his co-workers have shown, in human subjects suffering from this disease, that the splenic artery contains many times more spherocytes than the splenic vein and that the splenic pulp spaces are packed with these abnormal erythrocytes.

Hematological Effects of Splenectomy

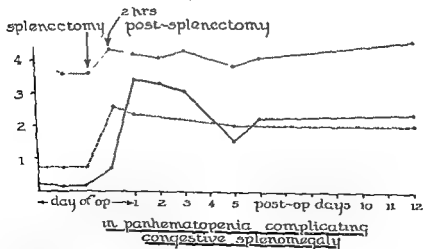
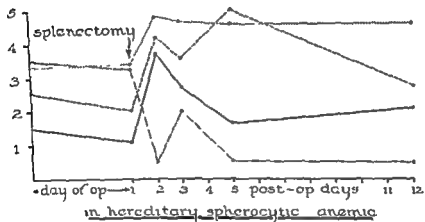
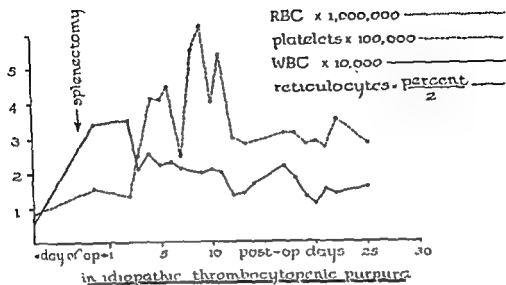


Fig 186.—Hematological effects of splenectomy in
 (a) idiopathic thrombocytopenic purpura,
 (b) hereditary spherocytic anemia,
 (c) splenic panhematopenia in congestive splenomegaly.

Clinical Features.—The onset may be explosive with gastrointestinal bleeding, cerebral hemorrhage, metrorrhagia, gross hematuria, or bleeding from the site of an extracted tooth. More often, however, it is insidious with a history of easy bruising, of frequent epistaxis, or a tendency to bleed from the gums. The onset in children is frequently preceded by some acute infectious process. Spontaneous petechiae of the skin and mucous membranes usually occur before massive hemorrhage. Large ecchymotic areas may appear after trivial injuries. Hemarthrosis is rare.

The commonest physical signs are those resulting from hemorrhage. An acute abdominal condition or an acute pleuritis may be simulated. Blood absorption produces fever. The spleen is never markedly enlarged and is palpable in less than one third of cases.

The course of the disease may be fulminating and rapidly fatal, or it may develop insidiously and exist for many years without severe manifestations, only to terminate with sudden cerebral hemorrhage. Spontaneous remission or recovery is common in childhood, uncommon after the age of 30 years.

Hematology.—Platelets are reduced and bleeding time is prolonged. Coagulation time is normal when determined by the usual methods, but the clot is friable and retracts poorly. A coagulation defect may be demonstrated by one of the prolonged clotting-time techniques or the prothrombin consumption test. The tourniquet test demonstrates that capillary fragility is increased. The leukocytes show no consistent abnormality. The degree of thrombocytopenia varies greatly. Bleeding usually occurs if the platelet count is below 60,000. Clinical bleeding, however, does not necessarily parallel the platelet count, especially after splenectomy, and bleeding may be absent with a platelet count of less than 60,000. Prolongation of the bleeding time is usually proportional to the degree of thrombocyto-

penia. The tourniquet test shows an increased number of petechiae below a blood pressure cuff which has occluded the venous but not the arterial circulation. After bleeding into the tissues has taken place, leukocytosis with a neutrophilia and a mild to moderate eosinophilia is not unusual.

The bone marrow findings are of great importance in excluding such conditions as leukemia, hypoplastic anemia, Gaucher's disease, or metastatic carcinoma, and in judging the probable effects of splenectomy. Megakaryocytes are normal or increased in number. It has been contended that the megakaryocytes exhibit immaturity and impaired platelet proliferation, and that they return to normal, both morphologically and functionally, after splenectomy.

Complications.—Bleeding is the chief complication. Its association with pregnancy deserves special mention, for 60 per cent of pregnant women with this disease do not survive. Mortality of the offspring is almost equally high. In a recent series, over 15% of the children were themselves purpuric. Splenectomy may be performed in pregnancy with resultant survival of the mother and birth of a normal baby.

Diagnosis.—Concealed hemorrhage may be difficult to diagnose. Bleeding into the kidney, the diaphragm, or the gastrointestinal tract may produce the symptoms of an acute surgical abdominal condition. Vaginal bleeding, due to purpura, must be differentiated from other causes. Intracranial hemorrhage may mimic a wide variety of neurological lesions. The differential diagnosis of the hemorrhagic diatheses must be considered. In this connection it is noteworthy that the clotting time may be normal in severe hypoprothrombinemia. Thrombocytopenic purpura must be distinguished from purpura without thrombocytopenia and also from those conditions in which the thrombocytopenia is a secondary manifestation. A rare and fatal cause of thrombocytopenic purpura is the widespread formation of platelet thrombi in capillaries.

and arterioles of females. Various drugs, such as Sedormid, quinidine, arsphenamine, and the sulfonamides may produce thrombocytopenia. If the condition is associated with leukopenia or anemia, out of proportion to the amount of blood loss, aplastic or hypoplastic anemia must be considered. If there is a significant degree of anemia, lymphadenopathy, splenomegaly, or hepatomegaly, the possibility of leukemia should be entertained. Liver disease and congestive splenomegaly should be excluded. Carcinomatosis, Gaucher's disease, Hodgkin's disease, and lymphosarcoma involving the bone marrow may induce secondary thrombocytopenic purpura. Allergic thrombocytopenic purpura rarely occurs.

Treatment.—Until very recently, splenectomy was the only effective therapy. During the past several years humoral antigen-antibody reactions have been demonstrated in patients who might otherwise have been classified as idiopathic thrombocytopenic purpura and may explain some of the operative failures. Thus, the Coomb's test is positive in a significant number of these cases and a hemolytic anemia may or may not be present. Transfusion of plasma from some patients with idiopathic thrombocytopenic purpura produces thrombocytopenia and purpura in normal recipients, and the life span of normal and polycythemic platelets is shortened when transfused into recipients having idiopathic thrombocytopenic purpura.

ACTH and cortisone have produced clinical and hematologic remission for variable periods in this disease. It is too early to state in what percentage of cases these drugs effect permanent remission. They are useful in pre- and post-splenectomy treatment but their beneficial effect is thought to be due to their stimulating effect upon platelet production and their depression of antibody production along the lines outlined above.

Blood transfusion has tided many patients over the acute phase of the disease and is usually essential in preoperative and post-

operative management. Because of the rapid disintegration of platelets in preserved blood, fresh blood is preferable. Eradication of focal infection, especially in children, may be followed by permanent remission.

For many years after the first reported successful case, splenectomy was considered to be contraindicated during an acute phase. More recently, however, it has become increasingly apparent that splenectomy may be lifesaving during an exacerbation. Improvements in operative technique and the more liberal use of blood transfusion have greatly reduced operative mortality, though splenectomy should certainly be withheld until the diagnosis is definitely established. Many today believe that medical management during the acute phase carries a higher mortality than operation. Some maintain that the chances of permanent remission following medical treatment are less than one in three.

Accessory spleens are present in about 25% of cases and probably cause one-third of the operative failures. As many as 400 accessory spleens have been reported in one patient. Their most common sites are the hilus of the spleen, the gastrosplenic omentum, the greater omentum, and the various peritoneal ligaments.

PRIMARY SPLENIC HEMATOPENIA

Definition.—An acute, chronic, or recurrent disease syndrome characterized principally by splenomegaly, normal or overactive bone marrow and varying degrees of anemia, neutropenia and thrombocytopenia.

The cause of these manifestations is said to be *hypersplenism*. It is thought that the spleen secretes a hormone which depresses maturation of normal cell components of the bone marrow and inhibits their delivery to the circulating blood and that, in addition, excessive destruction of the various cells occurs in the spleen. The syndrome, whatever its cause, is often cured by splenectomy.

Splenic hematopenia has been classified as primary or secondary. In the primary type,

no cause is detectable. In the secondary variety, such conditions as Hodgkin's disease, Gaucher's disease, cirrhosis of the liver and congestive splenomegaly may be etiologically significant.

Clinical Features.—These vary greatly and are largely dependent upon the relative degrees of anemia, neutropenia, and thrombocytopenia. The cellular components of the blood may be decreased singly, or in any combination. For example, anemia and neutropenia with normal thrombocytes may occur in one case, in another, thrombocytopenia and neutropenia may be present without anemia. Most commonly, all three components are decreased.

The course of the disease may be acute with high fever, agranulocytic angina, stomatitis, oral ulcerations, and occasionally ulcers of the lower extremities. Anemia and purpura may dominate the picture. Lassitude, weakness, low grades of fever and lowered resistance to infection are characteristic of chronic neutropenia. In some instances, neutropenia may recur in cycles. Occasionally, thrombocytopenia is associated with the menses. Anemia may or may not be hemolytic in character.

Hematological Findings.—Blood and bone marrow findings are obviously dependent upon which cell type is predominantly involved. If there is a hemolytic reaction, reticulocytosis, increased fragility, spherocytosis of red cells, elevated indirect serum bilirubin, and increased secretion of urobilinogen in stools and urine are common. If there is no acceleration of red cell destruction, or excessive bleeding, the anemia is usually normochromic and normocytic, without reticulocytosis or increased hemolysis. An essential diagnostic criterion is an active bone marrow with no abnormal cells.

Treatment.—Splenectomy is the treatment of choice. Blood transfusion may be required, and antibiotics are used to prevent and treat infection in the presence of neutropenia.

CHRONIC CONGESTIVE SPLENOMEGALY

The term splenic anemia was first introduced to describe anemia with splenomegaly not due to leukemia. In 1883, Banti recorded a syndrome of splenomegaly and anemia not associated with leukemia, Hodgkin's disease, malaria, syphilis, hemolytic icterus, or other recognizable pathological conditions. He noted cirrhotic changes in the liver, a chronic sclerosing endophlebitis of the splenic vein, and typical changes in the spleen itself. It is now recognized that elevation of splenic vein pressure, due to whatever cause, may produce a symptom-complex indistinguishable from that previously known as Banti's disease or syndrome and which is now most commonly referred to as *chronic congestive splenomegaly*. The majority believe that the etiology of this condition is an obstruction in the portal circulation, which may be either intra- or extrahepatic (see section on Portal Hypertension).

Clinical Features.—Splenic enlargement is usually marked. As noted by Banti, it is associated with a moderate anemia, which is normochromic and normocytic, unless complicated by hemorrhage or advanced hepatitis. A mild to marked leukopenia is more constant than anemia. Curiously, the relative percentages of all white cell types is unaffected by this numerical decrease. Thrombocytopenia is usually mild or moderate, but occasionally the platelets drop sufficiently to produce thrombocytopenic purpura. Bleeding from esophageal varices produces a posthemorrhagic anemia and masks typical hematological findings. In the presence of hepatitis the anemia becomes macrocytic. The disease usually appears before the age of 35, although it may develop either in infancy or old age.

Pathology.—In the youngest age group, the condition is commonly due to congenital anomalies. Although portal obstruction

is associated with about two-thirds of all cases of cirrhosis of the liver, no more than 20% of such patients develop congestive splenomegaly. It has been recorded that patients with Laennec's cirrhosis and congestive splenomegaly reveal more periportal fibrous tissue and distortion of blood vessels than cirrhotic patients without congestive splenomegaly.

The capsule and trabeculae of the spleen are thickened. Perisplenic adhesions are often abundant and highly vascularized. The pulp shows fibrotic changes. The Malpighian bodies are small and atrophic. Hyaline degeneration, periarterial hemorrhages, and hemosiderin nodules are found. The venous sinusoids are dilated. There may be chronic sclerosing endophlebitis of the splenic vein.

Since portal hypertension is presently accepted as the cause of congestive splenomegaly, problems of diagnosis and treatment will be included in the section on that subject.

PRIMARY NEOPLASMS OF THE SPLEEN

Neoplasms of the spleen are uncommon. The literature refers to 200-odd primary malignancies. Tumors may arise from the capsule and trabeculae, the lymphoid elements, endothelium or various reticulo-endothelial components. Primary carcinoma of the spleen is unknown, but metastatic carcinoma, especially from the lung and the breast, is present in about 2% of cases. Benign lesions, apart from their mechanical effects, are of little interest or importance. Twenty per cent of primary malignant tumors are lymphosarcomas.

Clinical Features.—The symptoms are those of a tumor in the left hypochondrium, inseparable from the spleen, associated with anemia, cachexia, local pressure effects, and radiological evidence of displacement of stomach, colon, and occasionally kidney. There may be ascites and pleural effusion.

Treatment.—Because of the rapid growth and metastasis of such lesions, early splenectomy is the only hopeful treatment. At best, survival seldom exceeds five years.

ANEURYSM OF THE SPLENIC ARTERY

Aneurysm of the splenic artery is rarely encountered. Operation has been recorded in about 60 cases of which 8 per cent were correctly diagnosed before operation. The commonest causes are arteriosclerosis and embolism. Symptoms are usually vague and inconstant. There may be dyspepsia and epigastric pain. A pulsating tumor is rarely palpable. Warning hemorrhage may produce an upper abdominal crisis requiring emergency surgery. Otherwise, fatal rupture is almost invariable. Removal of the spleen together with the involved vessel is generally successful, if undertaken before rupture has occurred. After rupture, the operative mortality thus far approaches 80 per cent.

CYSTIC DISEASE OF THE SPLEEN

Cysts of the spleen may be congenital or acquired. True cysts are dermoid, epidermoid, or endothelioid. False cysts are caused by trauma, infection or parasitic (hydatid) infestation. They may develop following the degeneration of an infarcted area. Splenomegaly due to any cause is said to be an etiological factor. The tumor may produce symptoms because of its large size, local pressure effects, or irritation of overlying peritoneum. X-rays aid in diagnosis by demonstrating displacement of adjacent organs. The treatment is splenectomy. Incision and drainage or marsupialization are reserved for complicated hydatid disease, when splenectomy is impossible. Enucleation of the cyst is no longer practiced.

ABSCESS OF THE SPLEEN

Abscess of the spleen is infrequent in temperate climates, but may complicate the course of any acute specific infection, such

as typhoid fever, pyemia, bacterial endocarditis, and parasitic infestations (hydatid disease, malaria and amebic dysentery). It sometimes follows trauma.

Clinical Features.—The signs and symptoms of splenic abscess are those of left subphrenic suppuration, but they may be delayed until a deeply placed focus has involved the peritoneal coat. Pain in the left upper abdomen, lower chest, and at times in the left supraclavicular region is continuous, severe, and aggravated by respiratory excursions. Fever is high and associated with chills. The patient is acutely ill, suffers from diarrhea and vomiting and rapidly loses weight. The abscess may penetrate into the general peritoneal cavity, into the subdiaphragmatic space and thence into the pleura, into any adjacent abdominal viscus, or outward through the abdominal wall.

Treatment.—Splenectomy, in conjunction with appropriate chemotherapy, is the best form of treatment. In the presence of extensive suppuration, however, one must be content with incision and drainage.

GRANULOMATOUS INFECTIONS OF THE SPLEEN

Splenectomy has been performed for granulomatous infections, such as tuberculosis and syphilis, apparently localized to the spleen. In the light of modern medical therapy, such operations should be rarely, if ever, necessary.

Where endemic, *malaria* is the commonest cause of splenic enlargement, and removal of such spleens was formerly practiced in tropical zones. At present, medical therapy controls the situation in the vast majority of cases. Kala-azar, rather than malaria, is probably responsible for the refractory exceptions. Emergency surgery, however, is not infrequently required because of rupture or torsion of a large malarial spleen.

Egyptian splenomegaly affords a classical example of Banti's syndrome produced by

increased portal venous pressure. In this instance, the obstructive factor is intrahepatic, caused by the ova of *Schistosoma mansoni* and the cirrhotic changes they induce. Splenic enlargement is of the congestive type, and associated blood changes are those of hypersplenism. Though not curative, splenectomy may delay the advance of this disease which, untreated, progresses to liver atrophy, ascites, and death usually within four years of onset.

GAUCHER'S DISEASE

Definition.—This is a chronic, familial disorder characterized by splenomegaly, hepatomegaly, and skeletal defects caused by accumulations of large pale cells (Gaucher's cells) containing the cerebroside kerosin.

Although familial, the disease rarely affects more than one generation and is most common among Jews. When the onset is within the first six months of life, death usually results before the end of the second year. Over half of the adult forms began in childhood.

Clinical Features.—Symptoms are chiefly related to bone involvement and to the mechanical disturbance caused by an enormous spleen. Invasion of the bone marrow by Gaucher's cells may cause such radiological changes as decalcification, compensatory sclerosis, and pathological fractures. Femora, vertebrae, and sternum are most frequently involved. The enlarged spleen may fill the abdomen. Being prone to infarction, it is frequently painful. The head, neck, and extremities often develop a brownish pigmentation. Wedge-shaped pingueculae and brownish pigmentation of the sclerae and conjunctivae may suggest the diagnosis.

Although the hematological findings are to some extent those of secondary hypersplenism, their essential cause is the replacement of normal bone marrow by Gaucher's cells. Unless thrombocytopenia causes hemorrhage, the anemia is normochromic and normocytic. Occasionally, it may be macro-

cytic due to the presence of nucleated red cells and reticulocytes. Thrombocytopenic purpura develops in over half of the cases. Leukopenia and neutropenia are frequent and are usually associated with a relative lymphocytosis.

The diagnosis is established by marrow or splenic aspiration yielding the large, pale, kersin-containing reticulum cells of Gaucher which, with Mallory's anilin blue stain, show numerous spider-like fibrillae.

Treatment.—There is no curative treatment. However, such patients suffer from the effects of hypersplenism as well as from the encumbrance of a massive intra-abdominal tumor, and these symptoms may be alleviated by splenectomy.

ACQUIRED HEMOLYTIC ANEMIA

Definition.—Acquired hemolytic anemia may be idiopathic, or it may occur in association with a variety of granulomatous, pyogenic or enteric infections, with Hodgkin's disease, lymphosarcoma, reticulum cell sarcoma, leukemia, Gaucher's disease, schistosomiasis, ovarian cysts, and carcinomatosis.

Clinical Features.—If not idiopathic, the hemolytic anemia may be masked by the symptoms and signs of the primary disease. Its onset may be rapid and quickly fatal, or it may be a chronic affair, with prolonged anemia and jaundice, recurring remissions and exacerbations. In contrast to hereditary hemolytic icterus, the patient is often more sick than jaundiced. Anemia and jaundice, with elevation of the indirect plasma bilirubin and increased output of fecal and urinary urobilinogen, are usually present. The red cells rarely exhibit increased fragility to hypotonicity, but may have in addition lowered resistance to mechanical agitation. The anemia is usually normocytic but may be macrocytic. Spherocytosis is present in some cases. Reticulocytosis, polychromatophilia, basophilia, and basophilic stippling reflect increased erythropoiesis. Autoagglutinins, pathological cold

agglutinins, atypical agglutinins and antibodies, both of the simple agglutinin variety and the incomplete or blocking variety, are often demonstrable. Recently, the Coomb's test for coating antibodies has been found positive in idiopathic spherocytic acquired hemolytic anemia. It is almost always negative in hereditary spherocytic anemia and, consequently, is useful in differentiating the two types. The test usually remains positive after splenectomy.

Treatment.—Removal of a dermoid or pseudomucinous cyst of the ovary, or cure of certain infections, has resulted in remission of the hemolytic anemia. Occasionally, both splenectomy and cure of the underlying condition are necessary. In idiopathic acquired hemolytic anemia, splenectomy may be desirable in selected cases. When beneficial, the effect is probably due to the removal of the organ in which abnormal red cells are predominantly destroyed. During the past two years ACTH and cortisone have been used successfully in the control of acquired hemolytic anemia, both idiopathic and secondary. Relapse usually occurs, however, upon discontinuance of this hormonal therapy. The principal usefulness of these drugs at present would seem to be in the preoperative and postoperative management of such cases and for temporary control of hemolytic anemia secondary to serious underlying disease such as leukemia, etc. In a few instances splenectomy plus ACTH or cortisone have controlled the hemolytic process after both drugs had failed. When the hemolytic process is severe and all medical measures have failed, splenectomy may save life. In the less severe, chronic variety, splenectomy should be considered only after an adequate trial of medical management, the demonstration of active erythropoiesis and the absence of primary bone marrow disease. A rise of red cells and hemoglobin following adrenalin injection affords an additional indication for splenectomy.

Transfusion should be used with caution in this disease, because of the probability of atypical antibodies in the patient's blood. The life span of transfused normal red cells is shortened in patients with acquired hemolytic anemia, but unaffected in the presence of hereditary hemolytic anemia. In some instances destruction is so rapid that circulatory collapse, oliguria, and uremia may result. Severe reactions may be caused by blood which nevertheless satisfies all known criteria of compatibility. It is safest to transfuse with saline suspensions of washed red cells.

SPLENOMEGALY OF INDETERMINABLE ETIOLOGY

Enlargement of the spleen, sufficient to be palpated beneath the costal margin, is noted on rare occasions without demonstrable cause. Blood and bone marrow changes are absent. There is no general or localized lymphadenopathy. Portal venous pressure is normal. Signs of bacterial infection or parasitic infestation are lacking. There is no gross metabolic disturbance.

In such cases, the presence of some obscure pathological process, localized to the spleen, cannot be excluded. For this reason, it is the opinion of some surgeons that splenectomy for splenomegaly alone deserves serious consideration.

Supplementary Splenectomy

Removal of the normal spleen may greatly facilitate major surgical procedures on the upper abdomen and thorax and at the same time render these operations more effectual, particularly when their purpose is the eradication of malignant disease. Block resection of the spleen, the greater and gastrohepatic omenta, together with the stomach, increases the scope of radical operation for gastric cancer. Esophagoduodenal anastomosis is performed much more easily and safely in the absence of the spleen. The

same applies to abdominal repairs of large diaphragmatic hernias, with or without incarceration of the spleen.

Technique of Splenectomy

When the spleen is sufficiently mobile to be lifted out of the abdominal incision, its removal is a technically simple and safe procedure. When it is bound down by dense, vascular adhesions, splenectomy may have to be abandoned and the operation consist of some palliative procedure such as ligation of the splenic artery or division of as much of the vascular pedicle as can be safely reached.

Preoperative Measures.—Facilities for blood transfusion should be available. The stomach is intubated and kept deflated throughout the procedure to permit better exposure. Optimum posture is obtained with an air cushion supporting the lower left ribs and flank and the table is tilted slightly to the right.

Operative Incision.—Most surgeons prefer a long, left paramedian, epigastric incision. However, in some hands the curved subcostal approach has been highly satisfactory, especially when the spleen is greatly enlarged and the subcostal angle markedly widened. In cases of suspected rupture, a midline epigastric incision may be advisable. It can be opened and closed more swiftly than the others and affords easier access to the right upper quadrant. The abdominothoracic approach, combining a transverse or left paramedian epigastric incision with an extension through the bed of the 8th rib or 8th intercostal space and the diaphragm, has been recommended for difficult splenectomies. Thoracotomy, however, increases the operative risk.

Removal of the Spleen.—As soon as the abdomen has been opened, thorough exploration must be carried out. Injury to other organs must be excluded in accident cases; complicating regional extensions in inflammatory disease; metastases in neoplasms;

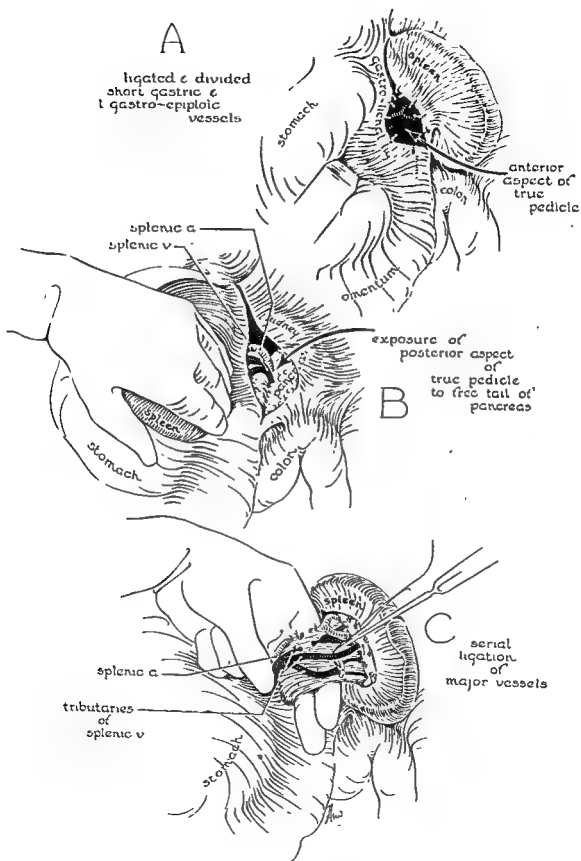


Fig 187.—Technique of splenectomy.

and calculous biliary disease in spherocytic jaundice. In this last condition and in thrombocytopenic purpura, accessory spleens must be removed. In congestive states, the site of portal obstruction should be accurately determined.

When the spleen is intact, free from adhesions and not too large, the following procedure is recommended. The lower two-thirds of the gastrosplenic ligament is divided between clamps. The spleen is tilted medially, the costal margin retracted, and the lienorenal ligament, together with its underlying areolar tissue, incised. The stomach and spleen are drawn downward and to the right to fully expose the very short upper third of the gastrosplenic ligament, which is then safely clamped and divided. When the avascular phrenocolical band is cut, the spleen is fully mobilized and may be lifted into the incision. The tail of the pancreas is carefully dissected away from the lower posterior portion of the pedicle. The splenic artery is divided between heavy ligatures. After a brief interval, to permit collapse of the spleen, the veins are individually ligated and divided and the specimen is removed. If the splenic reservoir is large, its evacuation may be hastened by the injection of a few minims of adrenalin into the artery just before it is tied. The pedicle stump is peritonealized and the roof of the lesser sac is reconstructed with a few interrupted sutures. The abdominal incision is closed in layers. Drainage is indicated in the presence of persistent bleeding, injury to the pancreas or established infection.

Perisplenic adhesions may be delicate and relatively bloodless, or dense and vascular. The former yield, as a rule safely, to manual exploration; but the latter have called a halt to many splenectomies. To cope with this type, the use of high frequency current has been recommended.

As a prelude to difficult splenectomies, it is sound policy to ligate the splenic artery

The vessel may be approached via the gastrophrenic omentum and tied close to its origin, or through the gastocolic omentum and secured where it parallels the superior border of the pancreas, several centimeters from the pedicle. Such a step will lessen blood loss and permit the patient to transfuse himself with the reservoir content of his spleen. Some feel that splenic artery ligation per se affords considerable palliation in cases of advanced cirrhosis that will not tolerate major vascular shunts. This we doubt (see Portal Hypertension).

Complications of Splenectomy

The complications of splenectomy may be the result of errors in technique, errors in judgment, and other causes relevant to certain types of splenopathy.

Technical errors may lead to postoperative hemorrhage, damage to adjacent viscera with subsequent perforation, peritonitis and fistula formation, infection consequent to local contamination and inadequate hemostasis, and wound disruption because of infection or faulty suture technique. The operation may be useless because of failure to remove accessory spleens. Expert hematological advice is the only safeguard against errors in the selection of cases for splenectomy.

In certain splenopathies, various postoperative complications are more commonly recorded. The removal of large spleens predisposes to pulmonary collapse and pneumonia. Temporary paralysis of the diaphragm occurs frequently. Vascular thrombosis and embolism are prone to follow splenectomy. The most common site of thrombosis is in the splenic vein. In this situation, it is thought to be the cause of the otherwise unexplained pyrexia that so frequently develops after the removal of congested spleens. Further extension of the clot into the portal vein is rapidly fatal. If adequate hemostasis is obtainable at operation, it is wise to administer anticoagulants

without delay. Debilitated individuals, with congested, adherent spleens, are particularly liable to develop left subdiaphragmatic suppuration. Hemorrhagic diatheses and malnutrition delay wound healing and pave the way for disruption. Closure, therefore, must be especially meticulous. Nonabsorbable suture material is preferable to catgut.

REFERENCES

- Cole, W. H., Walter, L., and Limarzi, L. R.: Indications and Results of Splenectomy, *Ann. Surg.* 129: 702, 1919.
- Dameshek, W., and Estren, S.: Symposium on Specific Methods of Treatment; Hypersplenism, *M. Clin. North America* 34: 1271-1289, Sept., 1950.
- Dameshek, W.: The Humors and Idiopathic Thrombocytopenic Purpura (Editorial), *Blood* 6: 954, 1951.
- Doan, C. A.: Hypersplenism, *Bull. New York Acad. Med.* 25: 625-650, 1949.
- Elliott, R. H. E.: Disorders of the Spleen With Special Reference to Those Amenable to Surgical Therapy, *Bull. New York Acad. Med.* 22: 415-427, 1946.
- Henry, A. K.: Removal of Large Spleens, *Brit. J. Surg.* 27: 464-474, 1940.
- Knisely, M. H.: Spleen Studies Microscopic Observations of Circulatory System of Living Unstimulated Mammalian Spleens, *Anat. Rec.* 65: 23-50, 1936.
- Lahey, F. H., and Norcross, J. W.: Splenectomy; When Is It Necessary? *Ann. Surg.* 128: 363-378, 1948.
- Lahey, F. H.: Technic of Splenectomy, *S. Clin. North America* 29: 739-745, 1949.
- MacKenzie, D. W., Jr., Whipple, A. O., and Wintersteiner, M. P.: Studies on Microscopic Anatomy and Physiology of Living Transilluminated Mammalian Spleens, *Am. J. Anat.* 68: 397-456, 1911.
- Maingot, R.: *Abdominal Operations*, ed. 2, New York, 1918, Appleton-Century-Crofts Company, Inc.
- Ponder, E.: Certain Hemolytic Mechanisms in Hemolytic Anemia, *Blood* 6: 559, 1951.
- Proceedings of the Second ACTH Conference, J. R. Mote, editor, Philadelphia, 1951, The Blakiston Company, vol. 2, p. 173.
- Proceedings of the Third International Congress of the International Society of Hematology, New York, 1951, Grune & Stratton, Inc. p. 91.
- Pugh, H. L.: Collective Review; Splenectomy With Special Reference to Its Historical Background; Indications and Rationale, and Comparison of Reported Mortality, *Internat. Abstr. Surg.* 83: 209-224, 1946.
- Rousselot, L. M.: Present Concepts in Surgery of the Spleen, *S. Clin. North America* 29: 369-382, 1949.
- Scott, R. B.: The Spleen and Splenectomy, *Brit. M. J.* 1: 1063-1070, 1949.
- Van Buren, G., and Curtis, G. M.: Principal Indications for Splenectomy During Childhood, *Arch. Surg.* 56: 125-131, 1948.
- Whipple, A. O.: Recent Studies in the Circulation of the Portal Bed and of the Spleen in Pathologic Conditions, *Ann. Surg.* 64: 1-14, 1916.
- Wright, O. H.: Splenectomy in Hemolytic Anemia, *Blood* 6: 195, 1951.
- Young, L. E., Miller, G., and Christian, R. M.: Clinical and Laboratory Observations in Autoimmune Hemolytic Disease, *Ann. Int. Med.* 34: 507, 1951.

CHAPTER XXIII

SURGERY OF PORTAL HYPERTENSION

DAVID W. MACKENZIE, JR., M.D.

INTRODUCTION

The conception that Banti's syndrome (see section on Spleen) is not a primary splenomegaly is not entirely new. Since the turn of the century various workers have considered the possibility of portal venous congestion producing the Banti picture of splenomegaly associated with anemia, leukopenia, thrombocytopenia and varying degrees of hepatic cirrhosis. In 1934, MacMichael coined the term *portal hypertension* and suggested that such a condition might occur in the absence of cirrhosis. In 1937, Whipple and Rousselot demonstrated manometrically many cases of greatly increased pressure in certain portal tributaries in patients with congestive splenomegaly whose livers were normal.

Though it is still occasionally stated that Banti's disease is caused by an intrinsic congestive derangement of the spleen, the majority are convinced by clinical and post-mortem experience that the essential etiological factor is an extrasplenic venous obstruction, situated at some proximal point in the portal bed or within the liver.

SURGICAL ASPECTS

Besides the hematopoietic consequences of congestive splenomegaly, portal hypertension is inevitably complicated by repeated and ultimately fatal hemorrhage from esophageal, gastric or other varices. Since splenectomy alone rarely cures more than the blood changes associated with so-called Banti's disease and affords little or no protection against hemorrhage, ways and means were sought by Whipple and his colleagues at the Columbia Presbyterian Medical Center of creating an adequate by-pass or shunt between a major portal tributary and a pe-

ripheral vein. Their efforts reached the stage of clinical application in 1943. In addition it was hoped that short-circuiting might lessen the rate of ascitic fluid accumulation, since such operations as omentopexy, saphenopertoneal anastomosis and the insertion of tubes between the peritoneum and outer layers of the abdominal wall had been without significant effect.*

The surgery of portal hypertension is, in most instances, palliative. Its fundamental indication is the control of gastrointestinal hemorrhage and, occasionally, of ascites. Though improved liver function in the cirrhotic group has been recorded frequently following successful shunting procedures, there is as yet insufficient evidence to suggest that the life expectancy of such individuals may be prolonged by surgery alone.

THE OBSTRUCTIVE FACTOR

In considering the surgical relief of portal hypertension, both the nature of the ob-

*Berman and Ruenhoff have recently published independent reports on ligation of the hepatic and splenic arteries in the treatment of portal hypertension resulting from cirrhosis of the liver. The rationale of this operation, first demonstrated (1907) in human cases by Herrick, is briefly as follows: (1) ligation of the splenic artery may diminish the total flow of portal blood by thirty or more per cent, and (2) ligation of the common hepatic artery, distal to its gastroduodenal branch, occludes hepatic arterial tension from the umbilical and capillary bed of the liver. In the presence of experimental or clinical cirrhosis, arterial pressure is transmitted to the portal vein almost seven times more readily than when the liver is normal.

With adequate chemotherapy, gas infection of the liver does not appear to complicate ligation of the hepatic artery.

trahepatic portal obstruction.

It is our belief that, in the presence of hypersplenism, splenectomy, in addition to the arterial ligations, should be done whenever practical. In our own small experience with

structive factor and its situation must be clarified. Rousselot has divided the obstructions into two major groups, intrahepatic and extrahepatic, as follows:

1. Intrahepatic

(a) Cirrhosis of the liver

- (i) Laennec's cirrhosis
- (ii) Schistosomiasis
- (iii) Biliary cirrhosis
- (iv) Infectious hepatitis

(b) Compression of vein

- (i) Inflammatory (e.g., pancreatitis)
- (ii) Pancreatic cyst
- (iii) Tumor
- (iv) Aneurysm of splenic artery

(c) Thrombosis of vein

- (i) Inflammatory
- (ii) Traumatic

(d) Cavernomatous transformation of vein (portal)

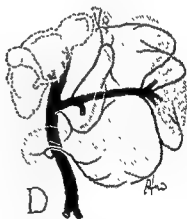
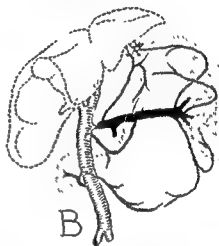
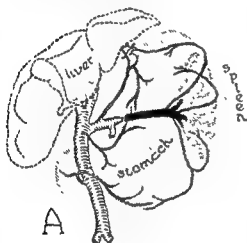


Fig. 188—Sites of obstruction causing increased hydrostatic pressure in the portal venous bed:

- (A) Obstruction of the splenic vein distal to the superior mesenteric vein.
- (B) Obstruction of the splenic vein proximal to the mesenteric and coronary veins
- (C) Obstruction of the portal vein.
- (D) Intrahepatic obstruction due to cirrhosis of the liver.

2. Extrahepatic

(a) Stenosis of vein

- (i) Congenital
- (ii) Acquired (phlebosclerosis)

DIAGNOSIS

The diagnosis of portal hypertension is usually easy. The spleen is enlarged. There is anemia, leukopenia, and often thrombocy-

topenia. There is frequently a history of hematemesis, and the presence of esophageal varices may be confirmed by radiological examination. If the block is intrahepatic, the liver is usually enlarged and its function tests abnormal. If the block is extrahepatic, the liver is normal, except in rare instances of purely coincidental cirrhosis. Diagnosis is more difficult and errors commoner in the absence of cirrhosis and hemorrhage. The nonspecific blood picture must be differentiated from other primary dyscrasias. Marrow biopsy is essential, and lymph node biopsy may also be indicated.

nary) and inferior mesenteric veins, which more often than not join the splenic vein before it enters the portal vein. At operation, therefore, it is essential to take venous pressure readings in several portal tributaries. For example, if pressure is elevated in the splenic vein, but normal in the superior mesenteric vein, the block is in the splenic vein and the portal vein is clear. If normal pressures are then found in known tributaries of the inferior mesenteric vein and of the left gastric (coronary) system, portal congestion is limited to the spleen and splenectomy will be curative. If

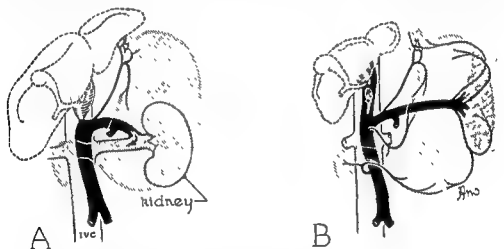


Fig 189—Standard shunting operations for the relief of portal hypertension

(A) Where the obstruction is in the portal vein and splenectomy with side-to-side anastomosis of the splenic to the left renal vein is carried out

(B) Anastomosis, side-to-side, of the portal vein and inferior vena cava, in the presence of intrahepatic portal obstruction

If the obstruction is extrahepatic, further localization is usually impossible before laparotomy. In a child, the commoner lesion is a cavernomatous transformation of the portal vein. In an adult, with a history of pancreatitis or of pancreatic cyst, obstruction of the splenic vein is probable. But, as a rule, accurate localization must await manometric pressure readings in various portal tributaries.

The only type of portal obstruction cured by splenectomy alone is situated in the splenic vein, distal to the left gastric (coro-

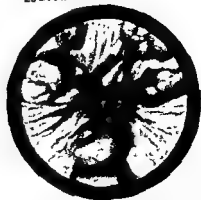
ny) and inferior mesenteric veins, which more often than not join the splenic vein before it enters the portal vein. At operation, therefore, it is essential to take venous pressure readings in several portal tributaries. For example, if pressure is elevated in the splenic vein, but normal in the superior mesenteric vein, the block is in the splenic vein and the portal vein is clear. If normal pressures are then found in known tributaries of the inferior mesenteric vein and of the left gastric (coronary) system, portal congestion is limited to the spleen and splenectomy will be curative. If

CORRECTIVE OPERATIONS

The short-circuiting procedures thus far attempted have been as follows:

1. Splenectomy with left nephrectomy and end-to-end splenorenal anastomosis
2. Splenectomy without nephrectomy and end-to-side anastomosis of the splenic to the renal vein.

ESOPHAGOSCOPIC VIEW



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Plate XXIV.—Esophageal Varices.

F. Netter, M.D.

The x-ray clearly demonstrates irregular filling defects caused by distended veins in the lower third of this esophagus. The varicose condition, however, commonly extends to the level of the aortic arch or even higher.

3. The operation of so-called Eck fistula, namely, end-to-side or side-to-side anastomosis of the portal vein to the inferior vena cava. Splenectomy is not performed.

4. Postsplenectomy alternative shunts have included anastomoses between:

- (a) the superior mesenteric vein and the inferior vena cava
- (b) the inferior mesenteric vein and the left renal vein
- (c) the stump of the splenic vein to the inferior vena cava, and
- (d) the inferior mesenteric vein to the inferior vena cava.

CHOICE OF OPERATION

Splenorenal anastomosis without nephrectomy and the Eck fistula are the operations of choice. An Eck fistula cannot be done in the presence of an obstructing lesion of the portal vein. Except in such conditions and especially in the cirrhotic group, Blakemore prefers this operation on the grounds that it is technically easier than splenectomy and splenorenal anastomosis; that it provides a larger stoma, less likely to be closed by clot or cicatrization; that, in the presence of cirrhosis, portal pressure is likely to be higher than when the block is extrahepatic; and that recurrent gastrointestinal hemorrhage has recurred far less frequently following this operation. Rousselot believes that splenectomy should always be basic to any operation for the relief of portal hypertension. He is confident in the effectiveness of the smaller, splenorenal shunt and points out that splenectomy overcomes the hematological effects of hypersplenism besides reducing the total flow of portal blood by as much as 40 per cent.

SELECTION OF CASES

The selection of cases for portacaval shunts is easier when the block is extrahepatic. The indication for surgery is hemorrhage, usually from esophageal varices. Liver function is normal and ascites uncom-

mon. Once the diagnosis is established, a transfusion or two generally suffices to prepare the patient for operation.

Portal hypertension, complicating cirrhosis of the liver, is a much more serious matter. Fortunately, no more than 20 per cent of cirrhotics develop congestive splenomegaly. In such cases, the decision to operate or not to operate must be governed by the extent of liver damage and by the capacity of the liver to respond to medical treatment. Because of the desperate situation induced by repeated hemorrhages, those responsible must be prepared to accept greater than normal risks inherent in surgical intervention. Based on liver function tests alone, experience suggests the following criteria of operability:

- (a) a serum albumin level of over 35%
- (b) Bromsulphalein retention less than 35% after 30 minutes, and
- (c) lessening positivity of cephalin-cholesterol flocculation.

It is emphasized that it may take the cirrhotic patient many months of intensive treatment to attain a state of improvement compatible with operation.

Modern medical treatment has done much to lessen the incidence and severity of ascites complicating cirrhosis. But there remain certain cases, adequately treated, in which the ascites persists unabated. Two factors contribute essentially to these failures: excessive liver damage involving the production of albumin, and portal hypertension. When the serum albumin responds to an energetic dietetic regime, but ascites remains, portal hypertension will be suspected and likely proved by x-ray evidence of esophageal varices. Such cases are candidates for portacaval shunts.

RESULTS OF SURGERY

In the largest reported series of portacaval shunts (Blakemore) there was an over-all operative mortality of 17.8%. In 43 cases of extrahepatic block there were 3 deaths. In 117 cases of cirrhosis deaths were 21.9%.

Among the so-called "poor-risk" cirrhotics, however, the operative mortality approached 40%

Ascites was intractable in 27% of the cirrhotic group. Hemorrhage occurred in 14% of the entire series, but was of a minor nature in 4.5%. Of 130 cases, 30.3% survived operation for a period of 3 to 6 years.

REFERENCES

- Blakemore, Arthur H.: Portacaval Shunting for Portal Hypertension, *Surg Gyn Obst.* 94: 443-454, 1952
- Berman, J. K., Koenig, H., and Saint, W. K.: Ligation of the Hepatic and Splenic Arteries in the Treatment of Portal Hypertension, *Quart Bull Indiana Univ M Center* 12: 99, 1950
- Everson, T. C., and Cole, W. H.: Ligation of the Splenic Artery in Patients With Portal Hypertension, *Arch Surg* 56: 153-160, 1918
- Herrick, F. C.: Experimental Study Into the Cause of Increased Portal Pressure in Portal Cirrhosis, *J. Exper Med* 9: 93, 1907.
- Moschcowitz, E.: Laennec Cirrhosis, Its Histogenesis, With Special Reference to the Role of Angiogenesis, *Arch Path* 45: 187, 1948
- Rienhoff, Wm F., Jr.: Ligation of the Hepatic and Splenic Arteries in the Treatment of Portal Hypertension, *Bull. Johns Hopkins Hosp.* 88: 368-375, 1951.
- Rousselot, L. M.: Combined (One-stage) Splenectomy and Portal Shunts in Portal Hypertension, *J. A. M. A* 140: 282, 1949
- Thompson, W. P., Caughay, J. L., Whipple, A. O., and Rousselot, L. M.: Splenic Vein Pressure in Congestive Splenomegaly (Banti's Syndrome), *J Clin Investigation* 16: 571, 1937
- Whipple, A. O.: Recent Studies in the Circulation of the Portal Bed and of the Spleen in Relation to Splenomegaly. *Tr. & Stud. Coll. Physicians, Philadelphia* 8: 203-217, 1941.
- Whipple, A. O.: Problem of Portal Hypertension in Relation to Hepato-splenopathies, *Ann Surg* 122: 449-475, 1945

CHAPTER XXIV

THE PERITONEUM, OMENTA AND MESENTERIES

J. R. MCCORRISTON, M.D.

INTRODUCTION

The peritoneum is the lining of the abdominal cavity. It is a serous membrane, derived from the mesenchyme, with a surface area approximately that of the skin. Since the peritoneum is in intimate contact with the intra-abdominal organs, the retroperitoneal tissues, the diaphragm and the abdominal parietes, it is not surprising that it is involved in a wide variety of disease processes, the majority of which have their origin in other tissues and organs.

EMBRYOLOGY

In the embryo, after the somatopleure and the splanchnopleure develop, the mesoderm splits laterally into the dorsal somatic mesoderm and the ventral splanchnic mesoderm. The cavity thus formed is the body cavity or celom which, eventually, is divided into the pericardial, pleural and peritoneal cavities. Variations may occur in the development of the peritoneum and mesenteries, but these are so numerous that no attempt will be made to describe each one separately. It is sufficient to realize that variations can and do occur with surprising frequency. Certain variations may be present throughout life without causing symptoms, whereas others may lead to serious conditions such as internal hernia, volvulus, or intestinal obstruction.

ANATOMY

In the male the peritoneum forms a closed sac, but in the female the free ends of the Fallopian tubes open into the peritoneal cavity. The mesothelial cells which form the peritoneal surface are flattened and polygonal, separated by cement substance.

This layer of cells rests on a thin layer of fibrous tissue, which is connected to underlying fascia or viscera by areolar tissue.

The continuous peritoneal membrane is invaginated by the hollow and solid viscera. This portion constitutes the visceral peritoneum, while the remainder is called the parietal peritoneum. The mesenteries of the bowel are formed by the two layers of invaginated peritoneum and the greater omentum is an extensive fold hanging from the greater curvature of the stomach. The peritoneal cavity is between the parietal and visceral peritoneum and contains, normally, only a small amount of serous fluid which lubricates its lining. The heavy folds of peritoneum attached to solid organs constitute their ligaments.

Anatomically, the peritoneal cavity is divided into two major compartments, the greater and lesser sacs, which communicate by means of the epiploic foramen (foramen of Winslow).

From a surgical point of view, there are four main divisions:

1. Supracolic.
2. Right infracolic
3. Left infracolic
4. Pelvic.

Surgically, the supracolic subdivision may be looked upon as the subphrenic space, as it lies between the diaphragm above and the transverse colon and its mesocolon below. This region is divided by the liver into supra- and infrahepatic portions. Three suprahepatic and three infrahepatic spaces exist. The falciform ligament divides the suprahepatic space into right and left parts. On the right side the right triangular ligament of the liver separates a large anterior space from a small posterior space. The

left triangular ligament extends along the posterior border of the left lobe of the liver separating the superior from the inferior surface. Therefore, only one space exists to the left of the falciform ligament

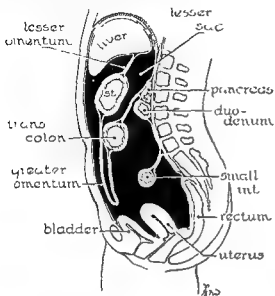


Fig 190—The peritoneum (median sagittal section)

This drawing illustrates, diagrammatically, the relationships of the omenta, transverse mesocolon, and mesentery of the small intestine

The three suprahepatic spaces are named

- (a) right superior anterior subphrenic space,
- (b) right superior posterior subphrenic space,
- (c) left superior subphrenic space

The infrahepatic region is divided into right and left parts by the second (descending) part of the duodenum. To the right of the duodenum and below the liver is the space often called Morison's pouch. To the left of the duodenum the space is divided into anterior and posterior parts by the stomach and lesser omentum.

The three infrahepatic spaces are named

- (a) right inferior subphrenic space (Morison's pouch),
- (b) left inferior anterior subphrenic space,
- (c) left inferior posterior subphrenic space (lesser sac).

These six spaces are all intraperitoneal, but an extraperitoneal space exists which corresponds to the bare area of the liver.

The right infracolic is separated from the left infracolic subdivision of the peritoneal

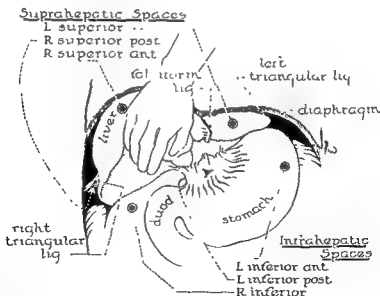


Fig 191—The subphrenic spaces

The six subphrenic spaces are shown from the anterior aspect. The right lobe of the liver is drawn medially and rotated upward.

cavity by the mesentery of the small bowel. The right and left paracolic gutters are important because they provide routes for the ascent and descent of exudate or pus. The pelvic subdivision is within the confines of the true bony pelvis.

FUNCTIONS AND REACTIONS

The peritoneal surface is smooth and moist, permitting parts of the visceral peritoneum to glide freely over each other and over the parietal peritoneum. The peritoneum has a marked capacity to absorb both fluids and small particles. Fluids which are not isotonic with body fluids are usually rendered isotonic before absorption takes place; then the rate of absorption may be rapid. The capillaries of the portal system are extremely permeable to protein molecules. Absorption of colloids by lymphatics and of crystalloids by capillaries is rapid and complete. Macrophages take up blood cells and particulate matter. The absorptive power of the greater omentum is enhanced by its mobility.

Under conditions of stress the peritoneal cells are probably capable of giving rise to macrophage cells. In any event the peritoneum has, at times, enormous powers of resistance to infection, which is exemplified by the fact that pure cultures of pathogenic bacteria introduced into the peritoneal cavity usually fail to produce peritonitis, unless there is severe traumatization or the introduction of foreign material. It has been shown that the powers of absorption and resistance are approximately equal in all parts of the peritoneum. It is probable that the peritoneum reacts as a whole in the presence of an infective process, although, grossly, there may be *localized*, *spreading*, *diffuse* or *generalized* peritonitis. When one area of the peritoneum is involved, the whole peritoneum pours forth exudate in its defensive effort.

The sensitivity of the peritoneum to a disease process is clinically important. The

visceral peritoneum has few or no nerves, but the bowel wall is supplied by the plexuses of Meissner and Auerbach (of the autonomic nervous system), while the parietal peritoneum is supplied by fibers of the cerebro-spinal system. The wall of the intestine may be burned, crushed or cut without producing pain, but traction or tension on many parts of the peritoneum is painful. The parietal peritoneum reacts to trauma as do other parts of the body supplied by cerebro-spinal nerves. Inflammations of the peritoneum which do not reach the parietal peritoneum may not cause pain.

The peritoneum has remarkable powers of repair, although the process is exactly the same as elsewhere in the body. After fibrous tissue forms in the wound it is covered with mesothelium and scarring is minimal. In the presence of severe trauma or infection, repair is by second intention; scar tissue is greater in amount, and adhesions or bands, which are often dangerous, may form. Such adhesions are primarily beneficial since they splint the parts, prevent or control leakage of exudate and permit complete healing. Once the acute phase is past, these adhesions tend to stretch, ultimately lose their blood supply and are absorbed. Although adhesions are necessary for repair, if there is much tissue trauma or extensive peritonitis, they may become dense, are not absorbed, and may lead to intestinal obstruction or lesser degrees of interference with function.

Mechanical and chemical injuries to the peritoneum are the commonest types. The injury may also initiate the reaction known as inflammation. If there is a peritoneal wound or tissue death, the process of repair will also be initiated.

The peritoneal fluid is normally small in amount, clear and viscid. It contains cells, over 90% of which are large mononuclear, polymorphonuclear and basophilic cells.

When an irritant is placed in the peritoneal cavity, polymorphonuclear leukocytes flood the region and become the predominant cells. Within a few hours mononuclear

left triangular ligament extends along the posterior border of the left lobe of the liver separating the superior from the inferior surface. Therefore, only one space exists to the left of the falciform ligament.

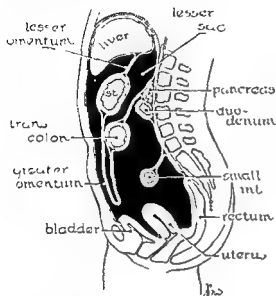


Fig 190—The peritoneum (median sagittal section)

This drawing illustrates, diagrammatically, the relationships of the omenta, transverse mesocolon, and mesentery of the small intestine

- The three suprahepatic spaces are named
- (a) right superior anterior subphrenic space,
 - (b) right superior posterior subphrenic space,
 - (c) left superior subphrenic space.

The infrahepatic region is divided into right and left parts by the second (descending) part of the duodenum. To the right of the duodenum and below the liver is the space often called Morison's pouch. To the left of the duodenum the space is divided into anterior and posterior parts by the stomach and lesser omentum.

- The three infrahepatic spaces are named
- (a) right inferior subphrenic space (Morison's pouch),
 - (b) left inferior anterior subphrenic space,
 - (c) left inferior posterior subphrenic space (lesser sac).

These six spaces are all intraperitoneal, but an extraperitoneal space exists which corresponds to the bare area of the liver.

The right infracolic is separated from the left infracolic subdivision of the peritoneal

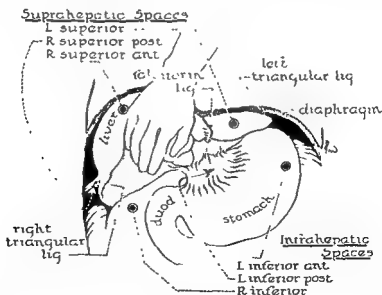


Fig 191—The subphrenic spaces

The six subphrenic spaces are shown from the anterior aspect. The right lobe of the liver is drawn medially and rotated upward.

cutaneous burn. Many general effects result from severe widespread peritonitis:

(a) Edema of the inflamed tissues pools body fluid which is initially at the expense of the extracellular fluid and later depletes the intracellular fluid. This is actually the creation of a third fluid space in addition to the intra- and extracellular spaces. As a result there are hemoconcentration, hypoproteinemia, hypochloremia and reduction in circulating blood volume. This, in turn, produces a degree of stagnant anoxemia.

(b) Bacteremia, toxemia and hemoconcentration all destroy erythrocytes, producing anemia.

(c) Peritoneal exudate and edema of the bowel wall inhibit peristalsis and so cause paralytic ileus. The concomitant distention exerts upward pressure on the diaphragm and secondarily interferes with pulmonary function.

(d) When obstruction of the bowel is caused by paralytic ileus, vomiting occurs, accelerating the process of dehydration.

(e) Dehydration and associated factors interfere with renal function, resulting in azotemia and oliguria or anuria.

All of these effects vary in degree of severity according to the suddenness of the inflammation and the treatment provided.

Diagnosis.—In the diagnosis of peritonitis, its presence, and then its source, must be determined. Initially, the symptoms and signs of peritonitis may not be distinguishable from those of the intra-abdominal lesion causing it. The fever is usually high, particularly so in children. Only in overwhelming infections is shock present and the temperature subnormal. The white blood cell count is high (e.g., 20,000 to 30,000 per c.mm.) with over 80% polymorphonuclear leukocytes present. The patient is distressed and concerned about his illness. He adopts a position in bed which reduces intra-abdominal tension to a minimum, i.e., his knees and hips are flexed and he breathes rapidly and shallowly. He tends to lie quite

still, for any movement causes an increase in discomfort owing to stretching or moving of the inflamed peritoneum and mesenteries. Examination of the abdomen will show some degree of distention, increased tone of abdominal muscles, and local or generalized tenderness with or without rebound pain. In the later stages of severe peritonitis marked abdominal distention, vomiting, oliguria, coated tongue, foul breath, spiking fever, restlessness with picking at the bed coverings, delirium, and finally coma occur. X-ray films of the abdomen will reveal dilated loops of bowel with fluid levels.

Conditions which may be confused with peritonitis, particularly early acute peritonitis, include pneumonia, pleurisy, influenza, renal colic, retroperitoneal hemorrhage and retroperitoneal tumors. Once the presence of peritonitis is diagnosed, there follows the task of determining its source. Sources of peritonitis are discussed elsewhere under the various intra-abdominal lesions.

Treatment.—The treatment of peritonitis includes:

(a) Prevention by means of prompt attention to its cause.

(b) Elimination of the focus of infection.

(c) Prevention and treatment of complications.

(d) Inhibition or destruction of the bacteria with chemotherapeutic and antibiotic agents when possible.

(e) Provision of early drainage of abscesses.

Although it is, at times, advisable to postpone operative treatment until the general condition of a very ill patient is improved, removal or drainage of the focus of infection is very important. Closure of a perforated peptic ulcer in inflamed appendix as used in this connection carried out with

Complications of numerous and dangerous the death of the patient

macrophages appear in large numbers in the increasing volume of peritoneal fluid which also contains fibrin. Foreign particles or bacteria are caught in the meshes of clotted fibrin and phagocytosis occurs. As the days pass, the cells degenerate and, together with their phagocytosed contents, disappear. After several days, when the process subsides, the peritoneal fluid returns to normal. The preceding description is that of mild peritonitis which results in resolution, but all grades of severity of the reaction can occur, and if the infection is very intense, death may result from toxic absorption, dehydration, electrolyte imbalance, and other secondary factors. The inflammatory response of the peritoneum is shared by the underlying and surrounding tissues.

When the peritoneum is divided, or devitalized, repair follows. The process of inflammation is set up and fibrinous tissue fluid causes adhesions to form, which are at first fibrinous, but which may later become fibrous. The degree of development of adhesions depends upon many factors, not the least of which is the varying capacity of individuals to form adhesions. The factors which influence the formation and disappearance of adhesions are not understood so that various methods for their prevention have rarely proved successful. Gentle operative technique, removal of blood and foreign materials, and reduction of bacterial contamination to a minimum are the best means of avoiding persistent adhesions.

ACUTE PERITONITIS

Peritonitis is the term which denotes any inflammatory process in the peritoneum, regardless of its extent or severity. Peritonitis may be bacterial or chemical and it varies in extent from localized to generalized. Nonbacterial peritonitis follows the introduction of sterile traumatizing or irritating substances into the peritoneal cavity.

Body fluids such as bile or gastric juice, clotted blood, gauze, sulfonamide crystals, surgical instruments, talcum powder, and antiseptics all produce peritonitis with signs and symptoms indistinguishable from those of bacterial infection. More severe grades of peritonitis are caused by bacteria which are usually of intestinal origin. Pyogenic peritonitis is generally caused by one or a combination of two or more of the following bacteria: coliform bacteria, staphylococci, streptococci. Other organisms are frequently found, particularly anaerobes (e.g., gas-forming pathogens). Many of these organisms produce powerful toxins which seem to be responsible for the severe toxemia often found and which are more important as a cause of death than bacteremia.

SECONDARY PERITONITIS

Acute bacterial peritonitis follows *direct* contamination when bacteria reach the peritoneum secondarily from an intra-abdominal viscus, some adjacent tissue or organ, or through an abdominal wound.

Locally, in bacterial peritonitis, the peritoneum soon becomes covered with fibrinopurulent exudate and later pus. Such an exudate, bathing the bowel wall, causes decrease or loss of peristalsis leading to distention. The greater omentum may be successful in limiting the process to a localized peritonitis (or abscess), but if localization does not result, diffuse or generalized peritonitis ensues. When a relatively large focus of peritonitis is suddenly set up, absorption of toxic materials occurs over an extensive surface. The route of spread depends upon local anatomical structures, such as the paracolic gutters, gravitational spread into the pelvis or the watershed effect of the mesentery of the small bowel.

It has been pointed out that the surface area of the peritoneum is approximately that of the skin, and widespread peritonitis is, therefore, comparable to a very extensive

cutaneous burn. Many general effects result from severe widespread peritonitis:

(a) Edema of the inflamed tissues pools body fluid which is initially at the expense of the extracellular fluid and later depletes the intracellular fluid. This is actually the creation of a third fluid space in addition to the intra- and extracellular spaces. As a result there are hemoconcentration, hypoproteinemia, hypochloremia and reduction in circulating blood volume. This, in turn, produces a degree of stagnant anoxemia.

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(e) Dehydration and associated factors interfere with renal function, resulting in azotemia and oliguria or anuria.

All of these effects vary in degree of severity according to the suddenness of the inflammation and the treatment provided.

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(e) Provision of early drainage of abscesses.

Although it is, at times, advisable to postpone operative treatment until the general condition of a very ill patient is improved, removal or drainage of the focus of infection is very important. Closure of a perforated peptic ulcer or removal of an inflamed appendix are examples of procedures used in this connection, which should be carried out with minimal delay.

Complications of peritonitis may be both numerous and dangerous, and may lead to the death of the patient besides prolonging

the disease. Toxemia, ileus, pneumonia, actual organic bowel obstruction, and septicemia are all examples of grave complications of peritonitis.

Chemotherapeutic agents are employed to great advantage in the prevention and treatment of peritonitis, and their use does not interfere with other methods of treatment. When peritonitis is caused by an organism or organisms sensitive to these agents, they become indispensable. Vaccines are of no practical value.

In all cases of peritonitis close attention must be paid to the maintenance of fluid and electrolyte balance and the provision of blood transfusions when indicated. The intestines are kept at rest by means of continuous nasogastric or intestinal tube suction, morphine is used to allay fear, anxiety and pain, and nothing is permitted by mouth. Patency of the suction tube may be assured by irrigating it every two or four hours with 30 cc of physiologic saline solution, although this is not always necessary. Because production and absorption of vitamins in the alimentary tract is seriously interfered with in peritonitis, and because ordinary parenteral fluids contain no vitamins, parenteral vitamins, in high dosage, must be given daily.

PRIMARY PERITONITIS

Primary peritonitis is rare and may be looked upon as metastatic peritonitis, since the organism reach the peritoneum indirectly from an outside focus of infection. It may begin after pathogenic organisms reach the peritoneum via the Fallopian tubes, or via the lymphatics, but the common route is the blood stream. Organisms can usually be found in the blood in these cases, and often a respiratory infection is present beforehand. The lesions are typical of generalized peritonitis, the type of exudate present depending upon the causative organism. Primary peritonitis is usually caused by the streptococcus or pneumococcus. A less common causative organism is Friedlander's bacillus.

Pneumococcal Peritonitis.—Pneumococci reach the peritoneum via the blood stream from a focus in the respiratory tract, the accessory nasal sinuses, the middle ear or from the genital tract of female infants or children through the Fallopian tubes. The pneumococcus causes acute peritonitis and a thick, whitish exudate is formed. Cultures of peritoneal exudate yield pure pneumococcus.

Streptococcal Peritonitis.—This form of metastatic peritonitis leads to the production of a serosanguineous exudate which, on culture, yields pure streptococcus.

Acute primary peritonitis produces signs and symptoms of generalized peritonitis without localization. Foci of infection leading to primary peritonitis are hard to differentiate and may co-exist. For this reason surgical exploration is not infrequently indicated as soon as the patient's general condition permits. When the peritoneal cavity is opened, a culture and direct smear of the exudate should be made. If pure streptococcus or pneumococcus is found on examination of the smear, the abdominal wound should be closed without further traumatizing exploration. Treatment thereafter is conservative, consisting of general supportive and symptomatic measures combined with chemotherapy guided by sensitivity tests performed on the cultured causative organism.

LOCALIZED ABSCESES

Acute bacterial peritonitis tends either to resolve completely or to localize and form one or more abscesses. An abscess, in turn, may undergo resolution, may point toward the surface of the body and discharge its contents, or toward an abdominal viscus (e.g., rectum). At times such an abscess may not lead to any of the above but may become dormant for long periods of time, later resolving or flaring up to produce further tissue destruction. Not infrequently diffuse or generalized peritonitis becomes loculated so that multiple abscesses develop.

Abscesses develop in certain sites which warrant separate consideration since the conditions leading to them and their treatment differ.

Subphrenic Abscess.—In the section on Anatomy, the subphrenic spaces were considered in detail because of their importance in respect to abscesses which may develop within them. Subphrenic abscess is often

difficult to diagnose because of the paucity of local physical signs, although general signs and symptoms of severe infection may be present, with spiking fever, severe malaise, high leukocyte count and secondary anemia. When the superior subphrenic spaces or the bare area of the liver are sites of abscess formation, signs and symptoms of pleural and pulmonary involvement are usually present,



Fig 192.—Subphrenic abscess in right superior anterior space

This is a single x-ray film of the abdomen, with the patient erect, after the introduction of 1,500 c c. of oxygen into the peritoneal cavity. The right half of the diaphragm is elevated, inferior to it can be seen the gas shadow and fluid level in the abscess cavity. No oxygen has entered this subphrenic space because of inflammatory adhesions between liver and diaphragm. On the left side the oxygen of the induced pneumoperitoneum fills the left superior space, permitting the stomach and other organs to descend to a lower level.

whereas, if the inferior subphrenic spaces are involved, abdominal signs and symptoms predominate. A right superior anterior subphrenic space abscess, for example, often leads to pleural irritation, with the development of a pleural exudate and secondary lower lobe atelectasis. The abscess may penetrate the diaphragm and pleura producing empyema and lung abscess. A most important diagnostic aid in detecting or confirming the presence of a superior subphrenic space abscess is the use of fluoroscopy and x-ray films, with or without induced pneumoperitoneum. Owing to the presence of the subphrenic abscess the involved hemidiaphragm is elevated, relatively motionless on respiration, and the abscess may throw a soft-tissue shadow or displace the liver downward. When gas-forming organisms are present in the abscess, a fluid level will be seen with an overlying bubble of gas. When gas cannot be seen on x-ray examination, the failure of oxygen in an induced pneumoperitoneum to enter the superior subphrenic space is evidence of abnormal adherence of the serosa of the liver to the parietal peritoneum over the diaphragm.

Inferior subphrenic abscesses, if large, may be detected as abdominal masses, and x-ray examination (particularly with the aid of barium) may demonstrate displacement of the stomach or duodenum from its normal position.

Although the subphrenic spaces may become infected by organisms carried by the blood stream, the usual means of infection is the presence of infected material from an abdominal viscus (e.g., perforated peptic ulcer, gall bladder or appendix; abdominal bowel surgery; suppurative pancreatitis).

Pelvic Abscess.—Any type of bacterial pelvic peritonitis may lead to the formation of a pelvic abscess, although it may also result from the gravitational spread of infected material from any site in the abdomen. Pelvic abscess develops most commonly following pelvic inflammatory disease

in the female, suppurative appendicitis, and infective lesions of the large bowel. In addition to the general signs and symptoms of infection there are definite local signs and symptoms which permit the diagnosis to be made with certainty. Owing to irritation of the pelvic colon and rectum there is usually diarrhea, often accompanied by the passage of mucus in large amounts. Rectal examination will reveal the presence of a boggy, tender mass pressing against the rectum. If it is large, abdominal examination may reveal a tender mass rising out of the pelvis. Often a pelvic mass causes irritation of the bladder with symptoms of dysuria and frequency. In the female, pelvic examination will add to the information gained by rectal examination.

Paracolic Abscesses.—Abscesses develop frequently enough in the paracolic gutters and in the iliac fossae to warrant separate mention. On the right side lesions of the appendix and right colon, and on the left, lesions of the pelvic colon, are the common starting points of paracolic abscesses.

Treatment of Localized Abscesses.—In addition to conservative supportive measures and chemotherapy, localized abscesses require operative drainage in most instances. The indications for operative drainage include progression of the abscess, spread, pressure effects (e.g., pressure on the common bile duct of a right inferior subphrenic abscess), septicemia, intestinal obstruction, and failure to respond to conservative measures. In certain instances concomitant operative treatment of a causative lesion (e.g., previously undiagnosed perforated peptic ulcer) is necessary.

The surgical approach for drainage of right superior anterior subphrenic abscess is via a right subcostal incision with care to avoid opening the uninvolved peritoneal cavity. The right superior posterior subphrenic space may be drained by means of an extrapleural approach, through the bed of the 11th or 12th rib. The left superior

and inferior anterior subphrenic spaces may be drained through a left subcostal incision. The right inferior subphrenic space may be approached through a high right paramedian abdominal or subcostal incision, taking advantage, when possible, of previous post-operative adhesions to avoid contaminating the remainder of the peritoneal cavity. The left inferior posterior space (lesser sac) may be drained transperitoneally or retroperitoneally by means of an incision through the bed of the left 12th rib.

A pelvic abscess can usually be drained through the rectum or vagina, although it may be necessary to employ an abdominal route.

Right and left paracolic abscesses may be drained by means of incisions directly over them, with care to avoid opening the uninvolved peritoneal cavity.

The contents of abscesses should always be cultured, and chemotherapy guided by the type of organism present and its sensitivity to particular antibiotic agents. At times it is of great value to irrigate an abscess cavity at intervals with a suitable antibiotic in solution. When it is not feasible to provide dependent drainage, it is desirable to posture the patient to attain this end.

SPECIAL FORMS OF PERITONITIS

Tuberculous Peritonitis

Tuberculous peritonitis is more frequent in females than in males, and in the young than in the old. Frequently it is one manifestation of miliary disease, but may take origin from an abdominal organ. It often originates in the Fallopian tubes and in the appendix. There are two chief forms of tuberculous peritonitis:

1. The exudative type is a slowly developing disease which leads to the formation of ascites and small, grey tubercles studding the peritoneum.

2. The proliferative or dry type often goes on to the formation of areas of caseation and fistulous connections between loops

of bowel. External fistulas are apt to follow surgical procedures. Much granulation tissue develops forming matted masses in the abdomen, particularly in the pelvis. If tuberculous peritonitis is not associated with widespread tuberculosis elsewhere, the prognosis is fairly good.

Diagnosis.—In tuberculous peritonitis the abdomen is distended but not markedly tender on palpation. The onset is usually gradual or may be abrupt with signs and symptoms very similar to those of acute pyogenic peritonitis (see above). There are usually low grade fever, general malaise, loss of weight and some abdominal discomfort with a feeling of fullness. The presence of tuberculosis elsewhere in the body is a common finding. The white blood cell count tends to be normal or only slightly elevated, with a relative lymphocytosis and monocytosis.

Treatment.—The basis of treatment is general supportive measures and rest as for tuberculosis elsewhere. Streptomycin and para-amino salicylic acid have been used but an accurate evaluation of this form of therapy is not yet available. Foci of infection, such as the appendix or Fallopian tube, should be removed surgically. Strangely enough, simple laparotomy often benefits the patient.

Gonococcal Peritonitis

Gonococcal peritonitis arises secondary to infection of the female genitalia. It is rarely fatal, but is important because of the necessity to differentiate it from diffuse peritonitis of other origin. When the diagnosis is not clear, it is safer to explore the right lower quadrant and remove the appendix than to risk delay should the lesion prove to be acute appendicitis.

Treatment.—The treatment of acute gonococcal peritonitis is general supportive measures, bed rest, and penicillin in high dosage. Operative treatment is used only to drain persistent abscesses or to remove a chronic tubo-ovarian abscess (See chapter on Abdominal Aspects of Gynecology.)

whereas, if the inferior subphrenic spaces are involved, abdominal signs and symptoms predominate. A right superior anterior subphrenic space abscess, for example, often leads to pleural irritation, with the development of a pleural exudate and secondary lower lobe atelectasis. The abscess may penetrate the diaphragm and pleura producing empyema and lung abscess. A most important diagnostic aid in detecting or confirming the presence of a superior subphrenic space abscess is the use of fluoroscopy and x-ray films, with or without induced pneumoperitoneum. Owing to the presence of the subphrenic abscess the involved hemidiaphragm is elevated, relatively motionless on respiration, and the abscess may throw a soft-tissue shadow or displace the liver downward. When gas-forming organisms are present in the abscess, a fluid level will be seen with an overlying bubble of gas. When gas cannot be seen on x-ray examination, the failure of oxygen in an induced pneumoperitoneum to enter the superior subphrenic space is evidence of abnormal adherence of the serosa of the liver to the parietal peritoneum over the diaphragm.

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Miscellaneous Types

In acute rheumatic fever the peritoneum may become inflamed. *Actinomyces* may cause peritonitis, as may brucellae and lymphogranuloma venereum.

Chronic Peritonitis

It must be remembered that any type of acute peritonitis may become chronic, with thickening of the peritoneum and the formation of widespread, dense adhesions. When peritonitis lasts more than a few days, peritoneal reaction cells appear, which are probably phagocytic, and which resemble large mononuclear cells. Sections of such peritoneum show that these cells project from the surface. Quantities of cloudy peritoneal fluid often persist in chronic peritonitis, which, on culture, prove to be sterile. Such collections are often found in the vicinity of walled-off intraperitoneal abscesses.

Granulomas

In addition to chronic infective granulomas, such as tuberculosis and actinomycosis, the peritoneum may be involved in granulomatous processes caused by irritating foreign material.

Talc Granuloma.—Talcum powder as a surgical glove and instrument powder is now being replaced by other substances but is still in fairly widespread use. Talc crystals in contact with the peritoneum act as an irritant causing a foreign body reaction. Ordinarily this reaction subsides after a time and no harmful effects are noted clinically. However, in certain individuals, talc in the peritoneal cavity leads to a very pronounced reaction which is proliferative, producing widespread adhesions and masses of granulation tissue which resemble a slowly growing neoplasm. Harmful effects may appear when such masses become bulky and when widespread adhesions interfere with bowel function.

Treatment.—The obvious measure is prevention by using other types of surgical

glove powder, or by careful washing of the gloves and instruments before introducing them into the peritoneal cavity during an operation. Treatment of the established granuloma consists of relieving complications, such as intestinal obstruction due to adhesions, by appropriate surgical procedures. On a few occasions it has been possible to excise granulomatous masses.

Sulfonamide Crystal Granuloma.—Sulfonamide crystals, placed in the peritoneal cavity for the prevention and treatment of infection, occasionally cause a granulomatous reaction with widespread vicious adhesion formation. Crystalline sulfathiazole has been the worst offender because of its low solubility.

Granulomas Due to Nonabsorbable Suture Material.—A small foreign body granuloma forms about every nonabsorbable suture placed in the peritoneum as in other tissues. These reactions seldom reach harmful proportions unless secondary bacterial infection supervenes. If infection persists, sinuses often develop and persist until the suture material is discharged or removed surgically.

TUMORS AND CYSTS

Primary tumors of the peritoneum, omenta, and mesenteries are extremely rare. There is only one true primary tumor of the peritoneum, the so-called mesothelioma (endothelioma) which is diffusely infiltrating and malignant.

Cysts of the omenta and mesenteries are rare. Lymphatic cysts of the mesentery are considered to be congenital lymphangiomas. They are often single and vary from a few centimeters in diameter to a very large size. They have milky contents and a flat endothelial lining. Gas cysts are very rare, occurring in clusters along the mesentery of the small bowel. Hydatid cysts of the mesentery form multiple masses, sometimes of large size.

Metastatic malignant tumors (usually carcinomas) frequently involve the peritoneum. The three most common intra-abdominal primary sites are the stomach, large bowel, and ovary. Spread is direct by penetration of the serosa and dissemination over the serous surface. Implantations may appear in isolated places at a distance from the parent tumor. When the metastases are widespread throughout the peritoneal cavity, it is called abdominal carcinomatosis. Irritation of the peritoneum causes ascites to develop and the fluid is often sanguineous. Metastases from primary neoplasms at distant sites may occur in the peritoneum, carried by lymphatics, or, more commonly, in the blood stream (e.g., malignant melanoma).

Tumors arising in the retroperitoneal tissues, whether benign or malignant, are similar to tumors of connective tissue origin elsewhere. Retroperitoneal lipoma and sarcoma are the most common types.

It is noteworthy that peritoneal tumors are often very hard to diagnose by the usual means of examination, including x-ray methods. If there is free peritoneal fluid present, it should be aspirated and studied carefully with attention to: the type of cells present, bacterial culture, guinea pig inoculation for tubercle bacilli, direct smear, specific gravity, protein content and microscopic appearance of sections of the sediment.

Treatment.—There is no curative treatment for primary mesothelioma of the peritoneum. Paracentesis abdominis may be performed at intervals if a large volume of peritoneal fluid collects.

Cysts of the omenta and mesenteries require no treatment unless they become very large and cause symptoms. Benign cysts may be carefully dissected out of the mesentery, or if in the greater omentum, may be removed along with surrounding omentum.

Treatment of metastatic peritoneal tumors is limited to palliative operations to re-

lieve symptoms and interference with bowel function. X-ray therapy is of questionable value, except, perhaps, in the case of certain radiosensitive sarcomas.

Pseudomyxoma Peritonci

This curious condition may follow rupture of a pseudomucinous cyst of the ovary or of a mucocele of the appendix. Sometimes the entire peritoneal surface is studded with small tumor nodules which secrete an enormous amount of jelly-like material which accumulates in the peritoneal cavity. This material acts as an irritant producing a low-grade peritonitis with the formation of adhesions. The signs and symptoms include abdominal distention, pain, and interference with bowel function.

The treatment is removal of the primary focus, which occasionally results in the disappearance of the whole lesion, but the prognosis is usually poor. This condition must be differentiated microscopically from metastatic colloid carcinoma.

THE APPENDICES EPILOICAE

Significant lesions of the appendices epiploicae rarely occur, but torsion of an appendix epiploica may produce the clinical picture of an acute abdominal emergency. Occasionally the blood supply of an appendix epiploica is interfered with and it undergoes calcification, then appearing as a calcified shadow in x-ray films.

ASCITES

Ascites is an accumulation of serous fluid in the peritoneal cavity. The fluid may be a transudate or an exudate.

An *exudate* is a collection of fluid secondary to an inflammatory process and may be clear, turbid, or purulent, depending on the number of cells present. Blood may be present. The specific gravity is over 1.018 and the protein content over 30 grams per liter.

A *transudate* is a noninflammatory collection of fluid. Such fluid is clear, of light straw color, with a specific gravity below 1.018 and usually below 1.015. The cell count is low and the protein content under 30 grams per liter.

There are many causes of ascites. General causes include cardiac failure, renal disease, constrictive pericarditis and increased mediastinal venous pressure. Depletion of the plasma protein results in a decrease in plasma osmotic pressure and ascites. Locally, an increase in portal vein pressure from hepatic disease or obstruction of the portal system, an increase in lymphatic pressure from neoplastic or inflammatory obstruction of lymphatic vessels, or rarely an ovarian neoplasm (Meig's syndrome), can cause ascites.

Treatment of ascites may be divided into two broad categories: (1) conservative, and (2) operative treatment.

The latter includes such measures as paracentesis, insertion of peritoneal buttons, saphenofemoral anastomosis, venous grafts from peritoneal cavity to subcutaneous space, resection of parietal peritoneum, omentopexy, anastomosis of pelvic colon veins to saphenous vein, and portacaval shunts.

SPECIAL DIAGNOSTIC PROCEDURES

Peritoneoscopy

Peritoneoscopy is the procedure of visualizing the peritoneal cavity and its contents by means of an optical instrument. Rudnick has advocated peritoneoscopy in lieu of diagnostic laparotomy where it is necessary to determine malignancies, the extent of metastases, tumor masses, site of tumors, the character of the surfaces of viscera, or to confirm a diagnosis and to perform biopsies in certain instances. It is a minor procedure performed under local anesthesia with little risk. However, most surgeons favor laparotomy when it is necessary to inspect the peritoneal cavity and perform a biopsy.

Induced Pneumoperitoneum

Although *induced pneumoperitoneum* (which is the introduction of air or another gas, such as oxygen, into the peritoneal cavity) is used therapeutically, it is mentioned here merely to point out its value in diagnosis of intra-abdominal conditions. It has been used with considerable success in connection with the diagnosis and localization of subphrenic abscesses. Approximately 1,500 c.c. of oxygen are introduced into the peritoneal cavity of an adult without undue discomfort. By posturing the patient suitably the subphrenic spaces can be outlined by x-ray films, as can certain intra-abdominal organs and tumors. It is particularly valuable in outlining tumors of the internal female genitalia.

Auscultation of the Abdomen

Bowel sounds are produced by two chief factors.

1. Contracting muscles.
2. Onward passage of intestinal contents within the lumen of the bowel.

A large variety of sounds can be produced by these two factors.

The first duty of the student is to familiarize himself with normal abdominal sounds. To do this he must use his stethoscope every time he examines an abdomen, whether or not it is the site of disease.

Conditions which slow peristalsis have a tendency to make the sounds quieter. When no sounds are present, peristalsis is absent. Louder sounds usually accompany an increased rate of peristalsis or a peristaltic rush. The pathologic sounds can be grouped into three categories: (1) diminished, (2) increased, and (3) abnormal. Diminished sounds accompany diminished peristalsis, and in acute diffuse peritonitis the abdomen is silent. Those conditions which produce the quietest abdomen are: perforated peptic ulcer, acute pancreatitis, gunshot wounds of the abdomen, and diffuse pyogenic peritonitis (as from a rup-

tured appendix). An effusion diminishes the sounds. After abdominal surgery peristaltic sounds usually disappear for 48 hours or more, then they return and the patient experiences gas pains, which are a good omen. Bed rest, in itself, will result in diminished peristaltic sounds.

Increased sounds are found in incomplete mechanical bowel obstruction, spinal anesthesia, intraintestinal hemorrhage and following the ingestion of purgatives. In chronic intestinal obstruction the bowel musculature is hypertrophied and the sounds are more forceful and booming. If the obstruction becomes complete, the bowel becomes exhausted (atonic) and the sounds disappear, indicating that peritonitis has set in. Obstructive borborygmi are pathognomonic of mechanical obstruction and appear as mechanical tinkles which are resonating. This type of sound is produced because there is gas as well as fluid in the small bowel under these conditions. Often such borborygmi are easily heard in the patient's room without the aid of the stethoscope.

Abnormal sounds include respiratory vibrations, hepatic and splenic friction rubs, splashing sounds of pneumohydroperitoneum, bruit of an abdominal aneurysm, crepitation from subcutaneous emphysema, and rarely one may hear the "bruit de collision" of gallstones or renal stones knocking against each other. Only when the abdomen is distended are the respiratory and cardiac sounds transmitted through the abdomen. Various friction rubs may be heard when there is exudate on the parietal and visceral peritoneum.

REFERENCES

- Adams, H. D.: The Surgical Management of Perihepatic and Subdiaphragmatic Abscess, *S. Clin North America* 28: 685-692, June, 1948.
- Ladd, W. E., Botsford, T. W., and Curnan, E. C.: Primary Peritonitis in Infants and Children, *J. A. M. A.* 113: 1455-1459, 1939.
- Osborne, M. P.: Primary Friedländer-Bacillus Peritonitis, *New England J. Med.* 238: 361-364, 1948.
- Rudd, J. C.: *Peritonitis*, 1948.
- Thorpe, J. H.: *Peritonitis*, 1948.
- Wangensteen, O. H.: *Intestinal Obstructions*, ed 2, Springfield, Ill., 1942, Charles C Thomas.

CHAPTER XXV

INTESTINAL OBSTRUCTION

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INTRODUCTION

Intestinal obstruction is interference with normal progression of the contents of the intestine along its lumen. Obstruction may be due to many different causes, and the clinical picture produced is dependent upon many factors which require consideration when a case is evaluated. Intestinal obstruction may occur at one or more points, or in one or more segments of the bowel, from the beginning of the duodenum to the anal orifice.

APPLIED ANATOMY

There are five points in the intestinal tract where the lumen is normally narrowed

- 1 Pylorus (sphincter)
- 2 Duodenojejunal junction (ligament of Treitz)
- 3 Ileocecal valve (sphincter).
- 4 Junction of pelvic colon and rectum
- 5 Anal canal (sphincter ani)

The bowel wall is composed of several layers of tissue: mucosa, submucosa, circular muscle layer, longitudinal muscle layer, and serosa. In the rectum the circular layer of muscle is poorly developed, in the colon the longitudinal layer of muscle is grouped into three bands known as taeniae coli, and the retroperitoneal parts of the bowel and the lower rectum and anal canal are devoid of serosa.

The intestine is innervated by the autonomic nervous system which contributes to the plexuses of Meissner and Auerbach. The arterial blood supply of the greater part of the duodenum, jejunum, ileum and right colon is derived from the superior mesenteric artery; the remainder of the colon and most

of the rectum is supplied by the inferior mesenteric artery. The lower rectum and anal canal are supplied by the internal pudendal arteries. The veins of all of the small intestine, colon, and most of the rectum are tributaries of the portal system; those of the lower rectum and anal canal drain into the systemic veins. All of the lymph from the tract eventually passes through the cisterna chyli, although that of the anus goes first to the inguinal lymph nodes.

APPLIED PHYSIOLOGY

The chief functions of the intestine are: *secretion* of fluid and enzymes for the digestion of food, *absorption* of the products of digestion and *excretion* of certain waste materials. Normally, in the adult, about 4,000 c.c. of fluid are secreted proximal to the pylorus in each 24 hours (1,500 c.c. saliva, 2,500 c.c. gastric secretion), and beyond the pylorus another 4,200 c.c. are secreted (500 c.c. bile, 700 c.c. pancreatic secretion, 3,000 c.c. succus entericus). Reabsorption is such that only 100 to 150 c.c. of water are lost daily in the stool.

All of the gastrointestinal tract secretions are electrolyte-containing fluids which are approximately isotonic with the extracellular fluid, although individual ionic concentrations vary considerably from one point in the tract to another. Lockwood and Randall studied the composition of the contents of the gastrointestinal tract at different levels in respect to sodium, potassium and chloride concentrations and showed in tabular form the marked differences between one level and another.

This information is of vital importance when replacement therapy is estimated in a case of intestinal obstruction.

TABLE IX

| <i>*Gastrointestinal Tract Losses (in meq./L.)</i> | | | |
|--|----------------------|------------------|-------------------|
| | Na | K | Cl |
| GASTRIC (fasting) | 60.4 9-116 | 9.2 0.5-32.5 | 81.0 7.8-154.5 |
| SMALL BOWEL (suction) | 111.3 82-147.9 | 4.6 2.3-8.0 | 104.2 43-107 |
| ILEOSTOMY (recent) | 129.4 105.4-143.7 | 11.2 5.9-29.3 | 116.2 90-136.4 |
| ILEOSTOMY (adapted) | 46 | 3.0 | 21.4 |
| CECOSTOMY | 52.5 | 7.9 | 42.5 |
| <i>*Bile and Pancreatic Fistulas (in meq./L.)</i> | | | |
| | Na | K | Cl |
| BILE | 148.9 131-161 | 4.98 2.6-12 | 100.6 89-117.6 |
| PANCREAS | 141.1 113-153 | 4.6 2.6-7.4 | 76.6 54.1-95.2 |
| URINE—normal | 40-90 | 20-60 | 40-120 |

In order to carry out its function, the gastrointestinal tract propels its contents along its lumen by means of peristalsis. There are several types of peristalsis in the small intestine, including slow, gentle waves which transfer food masses, "peristaltic rushes" which pass along rapidly for variable distances, and antiperistaltic waves, which have been noted in the duodenum and in the terminal portion of the ileum. Other movements, which do not move masses of intestinal contents either orally or aborally, have been described as segmenting and pendular movements, and these serve only to mix the intestinal contents. Any interference with normal peristalsis can lead to serious derangements of fluid and electrolyte balance in addition to the local effects of the obstructive lesion upon the bowel

CLASSIFICATION OF OBSTRUCTIONS

For clinical purposes intestinal obstructions are conveniently classified according to cause (Wangenstein).

I Mechanical

(A) Narrowing of lumen.

- (1) *Structures of bowel wall:* Congenital (atresia, imperforate

anus). Acquired (inflammatory, traumatic, neoplastic).

- (2) *Obturation* (foreign body, gallstone).

- (3) *Compression from without* (tumor, cyst).

(B) *Adhesive bands.*

(congenital, inflammatory, traumatic, neoplastic).

(C) *Hernia* (external, internal).

(D) *Volvulus* (small bowel, cecum, pelvic colon)

(E) *Intussusception* (ileocecal, enterenteric, colocolic).

(F) *Errors in development of the intestine* (stenosis, reduplication)

II. Nervous.

(A) *Inhibition ileus* (paralytic, adynamic)

(B) *Spastic ileus* (dynamic).

III Vascular (thrombosis and embolism of mesenteric vessels).

PHYSIOPATHOLOGY

In acute obstruction of the small bowel, the intestine above the site of obstruction becomes more and more distended by fluid and gas in direct proportion to the length

of time which has elapsed. These effects are maximal immediately above the point of obstruction, but the whole proximal tract can become involved if there is no relief.

It has been demonstrated that a pressure of 55 to 65 mm of mercury within the small bowel causes compression of veins which encircle the gut, interferes with absorption of fluid and gas, and leads to more distention. As distention and intraluminal pressure increase, the arterial flow is gradually obstructed, resulting in ischemia of the bowel wall. Gas in distended bowel is mainly nitrogen from swallowed air and partly carbon dioxide. Nitrogen makes up 60 to 80% and carbon dioxide about 4%. It has been noted that the normal small intestine is capable of absorbing 2,500 c.c. of carbon dioxide and 1,300 c.c. of other gases per hour, but in obstruction this ability is greatly reduced because of edema and partial ischemia of the bowel wall. Distention of the bowel with attendant edema and ischemia of its wall leads to inflammatory changes and ulceration. "Weeping" of the mucosa takes place owing to increased capillary permeability and venous pressure, adding to the fluid content of the bowel. In addition to the fluid and gas accumulating above the site of obstruction, large volumes of fluid escape into the lumen from the abnormal mucosa. As a result of this loss of fluid from the body fluid compartments, chemical changes appear in the fluids and tissues.

If untreated, these progress, resulting in dehydration, hypochloremia, acid-base imbalance, hypoproteinemia, hypovitaminosis, hemoconcentration and azotemia.

Acute obstruction of the large bowel, however, rarely produces such marked chemical imbalance. This is true, particularly in the earlier stages of obstruction, because the function of the small intestine is not impaired and most of the fluid and gas passing along its lumen is absorbed before reaching the large bowel. Later, however, when distention of the colon proximal to the point

of obstruction has developed sufficiently to raise the intraluminal pressure in the lower ileum, the physiological functions of the small bowel are impaired and the pathological changes of small bowel are added to those of large bowel obstruction.

In the case of large bowel obstruction, when the ileocecal valve is competent, a condition known as "closed loop" obstruction develops. The closed loop extends from the ileocecal valve to the site of obstruction, and distention is often limited to this segment of colon, with little or no concurrent distention of the small intestine until relatively late. There is then danger of perforation of the cecum which is the thinnest and most distensible part of the large bowel.

Fluid in the obstructed small bowel is usually brownish, thin and flaky. The contents of obstructed colon, in addition to gas, are feces, which may be either solid or liquid. When strangulation is present the bowel contents contain blood.

Pathologically there are several varieties of intestinal obstructions, e.g., *high and low, acute and chronic, partial and complete, simple and strangulating, of large bowel and of small bowel*. There are, however, from a clinical viewpoint, two main pathological types:

(a) *Simple obstruction* (obstruction of intestinal continuity).

(b) *Strangulating obstruction* (obstruction of continuity with interference with the blood supply of the intestinal wall).

In the strangulating obstructions the unfavorable primary effects become manifest more quickly than those of simple obstructions because the viability of the intestinal wall is threatened from the onset.

DIAGNOSIS AND CLINICAL PICTURE

Intestinal colic, concurrent intestinal gurgling, and the general complaint of gas pains have usually been noted by the patient. In intestinal obstruction the obstruc-

tive triad of distention, obstipation, and vomiting is to be expected, although one or two of these findings may be absent. When simple mechanical obstruction develops, the onset of symptoms varies in its rapidity according to its level, being slower the lower the level of the lesion. When there is strangulation of a segment of bowel, the onset may be very acute with severe symptoms and marked signs developing very rapidly.

the symptoms and signs become worse until coma develops and death ensues. Laboratory studies in an advanced case reveal hemoconcentration, rising hematocrit, reduced blood volume, early hyperproteinemia with later hypoproteinemia, leukocytosis, rising serum nonprotein nitrogen, urine of high specific gravity, high serum potassium and low serum sodium and chloride levels, with acid-base imbalance.



Fig 193—Small bowel obstruction Single film of abdomen shows coils of small bowel grossly distended by fluid and gas.

The general findings in obstruction are not specific and vary with its type, site, and duration. In advanced cases they are those of rapidly increasing dehydration and toxemia, with thirst, dry coated tongue, foul breath, dry loose skin, soft sunken eyeballs, oliguria or anuria, rapid thready pulse, hypotension or frank shock, and signs of a failing peripheral circulation. If untreated

Intestinal obstruction is a symptom complex rather than a disease, hence the diagnosis of "intestinal obstruction" is, by itself, insufficient. Four points require clarification:

1. The Presence of Intestinal Obstruction.—The history given by the patient is of utmost importance, and associated findings, such as a tender, irreducible hernia

mass, may make the diagnosis obvious. It may be difficult to decide whether or not abdominal distention is present, but the patient can often assist the surgeon in this respect. Obstipation is usually present, but it is absent in incomplete obstruction where hyperperistalsis may actually produce diarrhea. One or two bowel movements may occur after the onset of complete obstruction, emptying the bowel distal to the site of obstruction. In advanced cases visible peristalsis may accompany increased peristaltic sounds.

obstruction it appears later. As a rule, the higher the obstruction, the more severe is the vomiting. The vomitus becomes feculent when the lower ileum is involved. In obstruction of the colon vomiting is sometimes absent although reflex vomiting may occur initially. If the ileocecal valve is competent, vomiting is less pronounced, but if it is not, regurgitation into the small bowel occurs and copious vomiting ensues. X-ray films of the abdomen are invaluable in revealing where the distended loops of bowel are and the extent of distention present.



Fig 194.—Volvulus of pelvic colon. Single film of abdomen shows enormous distention of pelvic colon which has undergone volvulus.

2. The Site of Obstruction.—The most important factor differentiating large from small bowel obstruction is vomiting. In small bowel obstruction vomiting appears early, is frequent and copious, and the symptoms have an abrupt onset; in large bowel

When the large bowel is obstructed, a barium enema x-ray examination is conclusive, usually clarifying both the site and the nature of the lesion.

3 The Degree of Obstruction.—When there is complete intestinal obstruction, no

flatus or feces is passed. If incomplete, some is expelled, especially if repeated enemas are given. It must be remembered that the first enema removes material distal to the obstructive lesion, but subsequent effectual enemas indicate incomplete obstruction. Repeated x-ray films will show whether or not gas is getting by the lesion. After evacuant enemas, x-ray evidence of gas in the colon proves that gas has come down from above the site of obstruction. Enemas should not be given in the presence of an inflammatory lesion because of the danger of disseminating infection. The lower 25 cm. of the intestinal tract are always available for direct inspection by means of the sigmoidoscope.

4. The Presence or Absence of Strangulation.—The question of the presence or absence of strangulation can usually be settled by an examination for abdominal tenderness, which is marked in strangulation. Peristalsis is inhibited or absent after a time and, therefore, intestinal colic frequently is not a prominent feature late in strangulating obstruction. There are two co-existing varieties in this type of obstruction

(a) Strangulating obstruction of the involved segment of intestine, and

(b) Simple obstruction proximal to the site of strangulation.

This is the usual situation, although occasionally there is little or no evidence of distention, even on x-ray examination. Vomiting is an important early symptom of strangulation and often a mass can be detected in the tender abdomen. Absence of distention in strangulating obstruction may be explained only on the basis that the blood supply of the involved segment of bowel is unpaired without a mechanical block of its lumen. In this case the obstructive lesion does not completely occlude the lumen, so that transport of gas and fluid through the strangulated segment is possible despite its loss of viability. The x-ray finding of fixation in position of a coil of bowel, dilated

by gas and fluid, is suggestive of strangulating obstruction. Because the peritoneum and mesenterics are often inflamed and tense (e.g., volvulus), change in position of the patient may, through increased tension, cause pain and intensify the vomiting. However, in late simple obstruction with gross distention, peritoneal exudate forms and is responsible for tenderness and rebound pain. A rising leukocyte count is suggestive of strangulating obstruction.

When the preceding four points are clarified, a rather precise diagnosis as to the presence, site and degree of obstruction as well as to the presence or absence of strangulation, can be made.

DIFFERENTIAL DIAGNOSIS

The differentiation of some forms of intestinal obstruction from acute inflammatory intra-abdominal disease, may, at times, be extremely difficult, and often they co-exist. It must be remembered that many inflammatory conditions may secondarily cause adynamic ileus even though the lumen of the bowel is not mechanically obstructed.

Among those conditions which must be differentiated are simple constipation, renal colic, biliary colic, acute pancreatitis, coronary occlusion, acute pulmonary conditions, enterocolitis, and food poisoning. The correct diagnosis can usually be made by means of a careful history, physical findings, and laboratory tests.

TREATMENT OF INTESTINAL OBSTRUCTION

(A) Conservative Treatment

Infusions of electrolyte-containing solutions are capable of correcting electrolyte and water imbalance. In all intestinal obstructions fluid is lost from the normal body fluid spaces, whether it be by vomiting, by collection of fluid in the lumen of the obstructed bowel, or in edematous tissues. Fluid and electrolyte replacement therapy

must be directed very carefully in order to prevent or correct imbalances which can develop rapidly.

Decompression of the bowel above the site of obstruction by means of suction applied to an indwelling tube is essential. An accurate record of the volume and composition of both the intake and output of the patient is necessary. These values should be entered in the record every 12 hours, and totals recorded for each 24-hour period.

priate fluids, improves the patient's general condition although it does not relieve the obstruction. In obstructions of the colon, dehydration and electrolyte imbalance are less prominent features than in the case of small bowel obstructions.

In strangulating obstructions involving a long segment of bowel, blood loss may be very great. In such cases blood transfusions are required to restore the circulating blood volume to normal, and they are the most im-

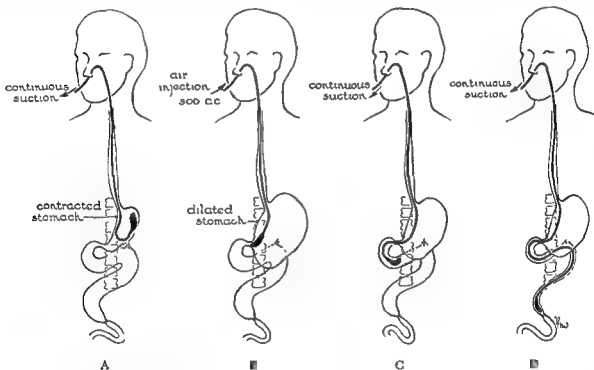


Fig 195—Technique of gastrointestinal intubation

Obstructive lesion of small bowel with distention of small bowel proximal to it. A Shows long mercury-weighted tube curled up in stomach. B 300 c.c. of air injected into stomach to permit tip of tube to progress freely to pylorus. C Shows tube passing down duodenum. D Tube has reached site of obstruction and proximal bowel is completely decompressed.

The urine, drainage fluids, and other excreta should be collected and representative samples analyzed for their electrolyte content. With accurate knowledge of the volume and composition of fluid lost, quantitative replacement is possible by choosing appropriate parenteral fluids. Saline solution is the most commonly used replacement fluid, and when used in combination with other appro-

portant conservative measures that can be carried out. Infusions of plasma are indicated in those instances when a large part of the circulating plasma has been lost by transudation into the peritoneal cavity. Because gross distention of the abdomen impedes the venous return from the lower extremities, it may be wise to employ the Trendelenburg position during the acute

phase of restorative therapy to facilitate venous return to the heart.

Inhalation of high concentrations of oxygen aid in eliminating the nitrogen of the air in the distended bowel by producing a steep partial pressure gradient. Its value is not very great for this purpose, but its value is considerable in the treatment of concomitant shock.

Conservative decompression of the bowel by means of indwelling tubes may be applied in all cases, but its use alone is contraindicated where *the possibility of strangulation is present*. Trials of conservative therapy even in simple obstructions often fail, leading to unwarranted delay of operative treatment.

There are two *absolute contraindications to the use of conservative decompression alone* in the treatment of obstruction:

- 1 Strangulating obstructions.
2. Obstruction of the colon with great distention.

By the same token they are *absolute indications for early operative treatment* because they are the two great items mainly responsible for mortality in cases of bowel obstruction. The general tendency now is for earlier operative intervention in those cases in which a reasonable trial of suction proves ineffective.

B. Operative Management

In early simple obstruction, operative intervention is indicated, after instituting suction decompression of the bowel and correcting dehydration and electrolyte imbalance.

In late simple obstruction the management is much more complicated. In this instance fluid and electrolyte imbalances have had time to develop to a serious degree. Vigorous fluid and electrolyte replacement therapy, with laboratory control, is essential, together with suction decompression of the distended bowel. Besides the above

measures, depleted blood volume must be bolstered and a determined attempt made to supply base-line caloric requirements.

In dealing with acute obstruction of the colon it is usually necessary to relieve the obstruction by means of a decompressive vent proximal to the point of obstruction. This vent may be made in the transverse colon (loop colostomy) or in the cecum (cecostomy).

Strangulating obstructions require very early surgical treatment and the bowel must be released from the mechanism which threatens its viability, or the bowel must be excised if nonviable. Usually it is possible to perform a resection and primary anastomosis in dealing with devitalized segments of small intestine; but in volvulus of the colon, exteriorization and excision of the devitalized bowel which has undergone torsion is the operative procedure of choice.

ADYNAMIC ILEUS

Adynamic, inhibition or paralytic ileus is a disorganization and impairment of the motor function of the gastrointestinal tract, sometimes resulting in complete cessation of motor activity with consequent obstruction.

Intestinal tone and peristalsis depend upon healthy intestinal musculature, unimpaired intrinsic nervous mechanism, balanced control of extrinsic innervation and physiological stimulation of propulsive activity. When these factors are impaired or absent, adynamic ileus results, and the degree of ileus depends upon how much interference with normal mechanisms exists. In established ileus there are sphincter spasm, disturbed propulsive motility and loss of intestinal tone.

Adynamic ileus may be complicated by mechanical obstruction, or ileus may complicate mechanical obstruction. Extensive ileus presents the picture of combined high and low obstructions with one type usually predominating.

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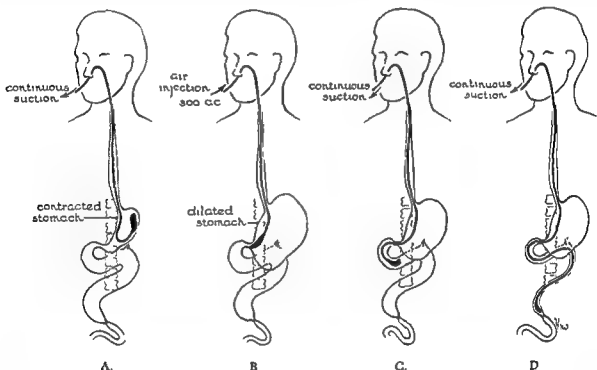


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priate conservative measures that can be carried out. Infusions of plasma are indicated in those instances when a large part of the circulating plasma has been lost by transudation into the peritoneal cavity. Because gross distention of the abdomen impedes the venous return from the lower extremities, it may be wise to employ the Trendelenburg position during the acute

phase of restorative therapy to facilitate venous return to the heart.

Inhalation of high concentrations of oxygen aid in eliminating the nitrogen of the air in the distended bowel by producing a steep partial pressure gradient. Its value is not very great for this purpose, but its value is considerable in the treatment of concomitant shock.

Conservative decompression of the bowel by means of indwelling tubes may be applied in all cases, but its use alone is contraindicated where *the possibility of strangulation is present*. Trials of conservative therapy even in simple obstructions often fail, leading to unwarranted delay of operative treatment.

There are two *absolute contraindications to the use of conservative decompression alone* in the treatment of obstruction:

- 1 Strangulating obstructions.
2. Obstruction of the colon with great distention.

By the same token they are *absolute indications for early operative treatment* because they are the two great items mainly responsible for mortality in cases of bowel obstruction. The general tendency now is for earlier operative intervention in those cases in which a reasonable trial of suction proves ineffective.

B. Operative Management

In early simple obstruction, operative intervention is indicated, after instituting suction decompression of the bowel and correcting dehydration and electrolyte imbalance.

In late simple obstruction the management is much more complicated. In this instance fluid and electrolyte imbalances have had time to develop to a serious degree. Vigorous fluid and electrolyte replacement therapy, with laboratory control, is essential, together with suction decompression of the distended bowel. Besides the above

measures, depleted blood volume must be bolstered and a determined attempt made to supply base-line caloric requirements.

In dealing with acute obstruction of the colon it is usually necessary to relieve the obstruction by means of a decompressive vent proximal to the point of obstruction. This vent may be made in the transverse colon (loop colostomy) or in the cecum (cecostomy).

Strangulating obstructions require very early surgical treatment and the bowel must be released from the mechanism which threatens its viability, or the bowel must be excised if nonviable. Usually it is possible to perform a resection and primary anastomosis in dealing with devitalized segments of small intestine; but in volvulus of the colon, exteriorization and excision of the devitalized bowel which has undergone torsion is the operative procedure of choice.

ADYNAMIC ILEUS

Adynamic, inhibition or paralytic ileus is a disorganization and impairment of the motor function of the gastrointestinal tract, sometimes resulting in complete cessation of motor activity with consequent obstruction.

Intestinal tone and peristalsis depend upon healthy intestinal musculature, unimpaired intrinsic nervous mechanism, balanced control of extrinsic innervation and physiological stimulation of propulsive activity. When these factors are impaired or absent, adynamic ileus results, and the degree of ileus depends upon how much interference with normal mechanisms exists. In established ileus there are sphincter spasm, disturbed propulsive motility and loss of *intestinal tone*.

Adynamic ileus may be complicated by mechanical obstruction, or ileus may complicate mechanical obstruction. Extensive ileus presents the picture of combined high and low obstructions with one type usually predominating.

Classification of Causes of Ileus

I *Intra-abdominal*

A. *Peritoneal irritation*

- (1) traumatic
- (2) bacterial peritonitis
- (3) chemical peritonitis

B *Vascular changes*

- (1) strangulation
- (2) mesenteric thrombosis

C *Extraperitoneal irritation*

- (1) hemorrhage
- (2) infection
- (3) renal lesions

II *Extra-abdominal*

A *Toxic*

- (1) pneumonia
- (2) uremia
- (3) empyema
- (4) systemic infection

B *Neurogenic*

- (1) lesions of spinal cord
- (2) lead poisoning
- (3) fracture of ribs

C *Electrolyte imbalance*

When adynamic ileus develops, peristalsis is greatly inhibited or absent, and the physiological upset is similar to that of organic obstruction without strangulation. Actual points of mechanical obstruction may appear secondarily where the greatly distended intestine is kinked upon itself. The pathological changes in the bowel wall resemble those proximal to an organic obstruction. Adynamic ileus is often present in cases of mechanical obstruction, particularly in advanced cases.

Clinical Picture of Adynamic Ileus.—In adynamic ileus there may be distention without colicky pain, but exudate in the peritoneal cavity later may cause abdominal tenderness. Vomiting begins and no gas is passed by rectum. The vomitus may become very copious, at first bile-stained and later feculent. Examination reveals anal sphincter spasm, distended small bowel and, sometimes, gas in the rectum. The abdomen is silent on auscultation. The diagnosis may be made by means of plain x-ray films of the abdomen before it is apparent clinically. In the films, gas appears before fluid, and

fluid levels appear still later. Distention has both local and general effects, as has been pointed out previously.

Postoperative ileus manifests itself in four main ways:

- (1) Post-anesthetic nausea and vomiting
- (2) Acute gastric dilatation.
- (3) Gas pains and temporary meteorism.
- (4) Established postoperative ileus.

Treatment.—(A) *Prophylaxis.* Postoperative ileus can usually be prevented by the following measures.

- (a) avoidance of preoperative cathartics,
- (b) gentle and aseptic operative technique,
- (c) avoidance of an excessive oral fluid intake in the first 12 postoperative hours, because sipping of fluids leads to air-swallowing,
- (d) avoidance of barbiturates and atropine immediately after operation, substituting morphine for them,
- (e) avoidance of drugs (e.g., Prostigmin) which stimulate the bowel during the first 3 or 4 postoperative days,
- (f) maintenance at all times of normal fluid and electrolyte balance,
- (g) gastric suction begun before major abdominal operations are performed.

B *Treatment of Established Paralytic Ileus.* Three general types of treatment of paralytic ileus have been used

1. *Aspiration of intestinal contents.* Gastric and intestinal intubation, with the application of suction to aspirate the contents of the distended gut for relief of the mechanical effects of severe distention.

2. *Resting of the bowel.* Nothing by mouth, morphine at intervals, and heat applied to the abdominal wall.

3. *Stimulation.* Repeated enemas, drugs (Prostigmin, acetylcholine, Pitressin, Pituitrin, apomorphine, bile salts, hypertonic saline introduced into the bowel lumen or vein, and cathartics).

Aspiration of bowel contents is the basis of treatment of paralytic ileus. During such

treatment close attention must be paid to the maintenance of fluid and electrolyte balance, of adequate potassium intake, of serum protein concentration, and of circulating blood volume. Very rarely, indeed, is it necessary to relieve the distention of bowel in paralytic ileus by establishing a jejunostomy under local anesthesia.

Stimulation of the bowel by means of drugs has but a very small place in the treatment of established paralytic ileus. During the phase of recovery, when disordered and uncoordinated movements appear, stimulative drugs are useless and may be harmful. Enemas appear to be mild stimulants of peristalsis during the phase of recovery.

REFERENCES

- Abbott, W. E.: A Review of the Present Concepts of Fluid Balance, *Am. J. M. Sc.* 211: 232, 1946.
- Devine, H.: A Concept of Paralytic Ileus: A Clinical Study. *Brit. J. Surg.* 34: 158-179, 1946.
- Gius, J. A., and Peterson, C. G.: Postoperative Ileus and Related Gastrointestinal Complications, *Internat. Abstr. Surg.* 79: 265-291, 1944.
- Lockwood, J. S., and Randall, H. T.: The Place of Electrolyte Studies in Surgical Patients, *Bull. New York Acad. Med.* 25: 228, 1949.
- Michel, M. L., Jr., Knapp, L., and Davidson, A.: Acute Intestinal Obstruction, *Surgery* 28: 90, 1950.
- Wangensteen, O. H.: *Intestinal Obstructions*, ed 2, Springfield, Ill., 1945, Charles C Thomas.
- Webster, D. R., Henrikson, H. W., and Currie, D. J.: The Effect of Potassium Deficiency on Intestinal Motility and Gastric Secretion, *Ann. Surg.* 132: 779, 1950.

CHAPTER XXVI

APPENDIX

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INTRODUCTION

The abdomen is opened more frequently for appendicitis than for any other abdominal condition. Therefore, a thorough knowledge of appendicitis, its various manifestations and complications, is necessary in order that proper treatment may be carried out. With improvements in diagnostic aids and the use of the antibiotics, there is less to fear from this condition by both doctor and patient. Paradoxically, this has resulted in earlier treatment, with lessening in the mortality. Fortunately, an emergency appendectomy is no longer performed for every pain in the right lower quadrant. The pendulum has swung to a more cautious approach, but when the diagnosis is certain, an immediate appendectomy should be carried out.

There are many references to inflammation of the appendix in the literature of the 19th century. Among the more important was a lecture delivered in Montreal by Dr. Howard in 1858, which was afterward published. "Clinical Lecture on Inflammation and Perforation of the Appendix Vermiformis" in the Montreal Medical Chronicle. The turning point in the history of the appendix is 1886 when Dr. Fitz of Boston coined the term appendicitis and advised early removal of the inflamed organ. In 1887, Thomas G. Morton, of Philadelphia, performed the first successful removal of the appendix. Sir Frederick Treves in 1888 added a refinement in technique by insisting on closure, rather than a mere ligature of the divided bowel. McBurney a year later wrote his classical paper describing the position of maximum tenderness in appendicitis, which is now known as "McBurney's point." Five years later Fowler described the tech-

nique of burying the stump of the appendix in the cecum. In the early part of the 20th century the expectant treatment was advocated by Ochsner and Sherrin.

ANATOMY

In human beings and higher apes the cecum terminates in a constricted wormlike appendage varying in length from 0.5 cm. to 33 cm., the average being 9 cm. with a diameter of 0.5 cm. This is called the vermiform appendix. The longitudinal taeniae of the cecum converge to form the external longitudinal muscle coat of the appendix.

The base of the appendix usually lies in the right iliac fossa, the surface marking of which is known as *McBurney's point*. This is at the junction of the middle and outer third of a line joining the right anterior-superior iliac spine to the umbilicus. With the cecum in the normal position, the appendix may be in front of or behind this portion of the bowel, or it may dip into the pelvis, or lie among the coils of small intestine. These four positions are important from the point of view of diagnosis and complications, such as appendiceal abscess. With congenital anomalies, such as non-descent of the cecum, the appendix may lie in the right hypochondrium under the liver. In malrotation of the gut, the appendix may be on the left side, and rarely, when there is a diaphragmatic hernia, the appendix may be in the left chest.

Structure of the Appendix

The structure of the appendix is similar to that of the rest of the bowel, consisting of serosa, longitudinal and circular muscles, submucosa and mucosa. The peritoneal

layer is closely adherent to the muscular coat except at the mesentery; the distal third, which is completely surrounded by adherent serosa, may be unable to expand when inflamed and this leads to early necrosis.

The appendix may be obscured by Jackson's membrane, which is a sheet of peritoneum extending from the lateral abdominal wall to the terminal ileum and covering the cecum and appendix. The surgeon, following the longitudinal bands or taeniae of the colon will come across the appendix at their convergence. The thickness of the submucosa varies greatly in different cases and consists of loose fibrous and elastic tissue forming a framework for nerves, blood and lymph vessels. Between the submucosa and the mucosa are nodes of lymphoid tissue made up of small round cells, lymphocytes, polymorphonuclear leukocytes and plasma cells which are indicative of inflammation and yet are normally present, so that the diagnosis of the more chronic forms is rendered difficult for the pathologist. The lymphoid tissue may contain a germinal center; hence the child's appendix is frequently called the "abdominal tonsil." The mucosa consists of a single layer of columnar epithelium and is arranged with pockets called the crypts of Lieberkühn. Between these crypts is the tunica propria containing connective tissue.

ETIOLOGY

Appendicitis is more common in males than in females. It may occur at any age, but is commonest from 10 to 30 years, and a familial susceptibility has been noticed. Nevertheless, no experimental evidence is available to prove the etiological factors concerned. At least it may be said that appendicitis is commoner in civilized communities, either due to a high protein diet or to a diminished content of cellulose, or both. Some factors, however, appear to have a definite responsibility in the causation of this condition. In early life the appendix

contains much lymphoid tissue which is especially prone to infection. The subsequent atrophy of this tissue may be the cause of the markedly reduced frequency in later years. There appears to be little doubt that fecaliths, obstructing the lumen and becoming increasingly inspissated, are an important etiological factor. This is most important in the appendicular colic described by Wilkie. Obstructive appendicitis due to a fecalith bears a remarkable similarity to diverticulitis. Such fecaliths cause obstruction of the lumen or erosion of the mucosa, followed by necrosis and perforation. This is very dangerous as it usually occurs before the inflammation has had time to wall off the area, and consequently widespread peritonitis is frequent. The infection may begin in the mucosa and is not necessarily due to irritation by the contents, as barium frequently remains in the appendix for several days without causing appendicitis. Kinking and distortion of the appendix by a short mesentery, tending to increase the possibility of obstruction, may also be a factor. Foreign bodies such as pinworms, seeds and indigestible food are an unusual and almost accidental cause of appendicitis. Anaerobic microorganisms may also play a part in the development of gangrene.

There is no evidence that the state of health of the patient plays a part in the etiology, as many healthy youngsters come to operation with all degrees of appendicitis and recover immediately when the offending organ is removed, unless complications delay the progress.

Recurrent attacks of appendicitis result in fibrosis with a narrowing of the lumen, and this may give rise to symptoms of partial obstruction. At times the obstruction may be complete, leading to obliterative appendicitis and occasionally to empyema of the appendix, or mucocele.

BACTERIOLOGY AND PATHOLOGY

The commonest organism cultured in cases of appendicitis is the colon bacillus.

However, infections are mostly mixed and the streptococcus plays an important role. In fact, it may be the primary factor. The improvement in many cases obtained by penicillin would appear to support this contention. In gangrenous appendicitis one of the anaerobic organisms, such as Welch's bacillus, may be a factor, as suggested by occasional postoperative infection in the abdominal wall.

Pathologically, the changes in appendicitis range from simple catarrhal inflammation to gangrene. A small area may show edema, round-celled infiltration, ulceration of the mucosa, suppurative inflammation or necrosis. On the other hand, the whole appendix may be involved in an acute inflammatory process, and be red, edematous, friable and gangrenous. An abscess may occur in the wall, or in the peri-appendicular tissue. A spreading peritonitis may be present with turbid fluid, or the infection may spread to the entire abdomen causing general peritonitis. The cecum may be involved and become infected, edematous, and friable, a point to be remembered when one considers inverting the base of the appendix in such cases. In an effort to wall off the infection, the omentum may wrap itself around the appendix and form a mass readily palpable on examination, but indistinguishable from a localized abscess. The small bowel may become adherent and give rise to symptoms of partial obstruction.

CLINICAL PICTURE

In considering the clinical picture of appendicitis, it will be easier if we divide it into acute, subacute, and chronic forms. The acute form should be divided into two fundamentally different types, the first of which is acute inflammation of the wall, and the second, acute obstruction of the lumen, or closed loop obstruction, which was originally described by the late Professor Wilkie of Edinburgh.

Acute Appendicitis

1. Acute Inflammation of the Wall—Acute Appendicitis.—Although diagnosis is very simple in a typical case of appendicitis, few diseases present so many diagnostic variants. The typical picture is one in which recent abdominal discomfort centers around the umbilicus, and is accompanied by nausea and, frequently, vomiting. Later the pain becomes localized in the right lower quadrant, with rebound tenderness, increased resistance and tenderness in the right iliac fossa, and tenderness on rectal examination, especially on the right side. The temperature is usually between 99° and 100°, with a leukocytosis of 10,000 to 14,000. If examination of the chest and the urine is negative, the diagnosis is established. The history is frequently of short duration, although there may have been previous attacks.

Diagnostic difficulties increase if the appendix is retrocecal, or if it lies along the lateral wall of the cecum, hangs over the brim of the pelvis, or lies adjacent to the ureter. Difficulties are further increased by the type of pathological change—catarrhal, acute fulminating, perforating, abscess forming, gangrenous or obstructive.

In children, the picture of appendicitis is considerably different from that of the adult. In apparently healthy children, crying and vague tenderness in the right iliac fossa may be the only signs of an acute appendicitis, so that extreme care in examination is necessary. On the other hand, this is the age of exanthemas, many of which have transitory abdominal pain. Gastroenteritis is also common during this period, and the best differential point is to administer sweetened fruit juices which act as a mild intestinal sedative in gastroenteritis but do not affect the pain of appendicitis. Fever may be unusually high, or the temperature may be normal. The leukocyte count is not of much help as it may be extremely high—20,000, or as low as 3,000 or 4,000.



In the aged, appendicitis is less common because of the gradual obliterative process which takes place during the years, but this may predispose to a closed loop obstruction and be all the more serious in elderly patients with low resistance. Here, the only safeguard is to remember appendicitis as a possible diagnosis.

The original pain in the epigastrium or around the umbilicus is usually of a cramp-like nature, but when it settles in the right lower quadrant, it is steady and is increased by movement or blowing out of the abdominal wall. Sudden release of pressure on the right side of the abdomen causes pain in the right iliac fossa, so-called rebound pain. This test should not be carried out too vigorously as it could conceivably break down recently formed adhesions and spread the infection. Rovsing's sign is frequently present—pressure over the large bowel causes pain in the right iliac fossa. Hyperesthesia is often found over the appendix. If the lumen of the appendix is obstructed, the pain is usually crampy, or colicky in nature, and in the early stages muscular resistance is unusual. When the appendix is retrocecal, all the signs are less marked, because it is shielded by the cecum. When the appendix lies in the lateral paracolic gutter, the pain and tenderness may be localized in the flank. If it is adjacent to the ureter, a few red blood cells may be found in the urine on microscopic examination.

Though the point of maximum tenderness is usually in the neighborhood of McBurney's point, this depends on the site of the appendix. The degree of tenderness and muscle spasm also depends on the position of the appendix, being much more marked when the organ lies against the parietal peritoneum of the right iliac fossa. Tenderness on rectal examination is more marked if the appendix is situated in the pelvis. If a mass is felt, it is due either to the presence of an abscess or to the omentum walling off the appendix as a protective mechanism. Fever will increase as the infection spreads,

with a rise in the pulse rate. When the patient is seen early in an attack, it is unwise to delay treatment because of the absence of fever or tachycardia.

Psoas spasm is a common sign, especially in retrocecal appendicitis. This is elicited by hyperextending the thigh or by flexing and adducting it against resistance. Obturator spasm may be present especially when the appendix lies in the pelvis, and can be noted by flexing the thigh and internally rotating the leg, which gives pain through spasm of the internal obturator muscle. While a history of constipation is frequently elicited, diarrhea may occur in appendicitis. As a rule, however, this suggests enteritis, and great care should be taken to establish the diagnosis before surgical intervention is carried out.

2. Acute Obstruction of the Lumen. Closed Loop Obstruction.—The main characteristic of the obstructive type is an intermittent, crampy pain which is very severe and made worse by movement. The cessation of this crampy pain is indicative of perforation, relieving the tension of the closed loop obstruction, but pain will recur in a few hours as a generalized peritonitis develops. In the early case of appendicular colic, which, as Wilkie states, so often goes on to perforation, it is usual to find a normal pulse and leukocyte count, but both of these will rise as soon as inflammatory changes develop, either in the wall or surrounding tissue. When leukocytosis is present, the differential count shows a marked increase in the polymorphonuclear cells with a shift to the left or to the more primitive forms.

DIFFERENTIAL DIAGNOSIS

A surgeon should consider the patient with an acute abdomen from two points of view: first, what is the most serious diagnosis, and second, are the signs and symptoms of such a nature that a laparotomy is advisable in spite of a failure to make a definite diagnosis. The latter observation especially applies to young children and the

aged. Many of the patients in these groups are left until perforation occurs because, until then, the signs are indefinite. In children, early pneumonia may simulate appendicitis, but the tenderness and spasm are less marked, the pain is frequently higher, the respiratory rate is increased and is frequently accompanied by movement of the abdominal wall and alae nasae. The fever, pulse rate, and leukocyte count are higher than in appendicitis.

In considering the differential diagnosis, it seems easier to classify the conditions into three groups:

A. Those associated with pain and protective muscular resistance.

B. Those with pain without muscular spasm.

C. Those associated with a mass in the right iliac fossa.

A. 1. *Acute Salpingitis*.—In the female, acute salpingitis frequently presents diagnostic difficulties. The most important diagnostic points are the history of exposure, a burning sensation on urination, a vaginal discharge, obtaining a drop of pus by milking the urethra, and marked tenderness on moving the cervix. The sedimentation rate usually shows a greater increase than in appendicitis. If the tenderness is bilateral on bimanual pelvic examination, a waiting policy with the use of antibiotics is advisable.

2. *Ruptured Ectopic Gestation*.—Internal hemorrhage and shock predominate, with softening of the cervix and moderate enlargement of the uterus. There is usually a history of a missed period.

3. *Early Pneumonia*.—Early pneumonia, especially of the basal type, not only in children but sometimes in adults, may cause confusion. Careful examination of the localization of the pain is necessary. In pneumonia it is usually higher in the abdomen and is associated with an increased respiratory rate.

4. *Perforated Peptic Ulcer*.—A perforated peptic ulcer usually gives a characteristic boardlike rigidity in the upper half of the abdomen, but if the gastric contents move down the right paracolic gutter, pain, tenderness, and even a mass may be found in the right iliac fossa. A history suggestive of a chronic ulcer may offer a clue. A plain film of the abdomen showing free gas under the diaphragm, along with an accurate history, should assure a correct diagnosis.

5. *Acute Cholecystitis*.—Acute gall bladder conditions may also simulate acute appendicitis. The appendix in cases of non-descent of the cecum may be adjacent to the gall bladder or the liver and the gall bladder may lie lower than usual. It is important to know whether the patient has had previous attacks of pain in the right hypochondrium. Radiation of pain to the angle of the scapula or to the back, or a history of flatulent indigestion, especially in a middle-aged obese female, suggests gall bladder disease. Further, in cases of acute cholecystitis, the globular tender surface of the gall bladder, moving with respiration, can usually be palpated, and a positive Murphy's sign is pathognomonic.

6. *Regional Ileitis*.—This may be confused with recurrent appendicitis. The long history of cramps and diarrhea, with gastrointestinal disturbances, suggests the advisability of having a barium series, with follow-through of the barium through the ileum, when the characteristic picture of "string and puddle" confirms the diagnosis. In the acute stage of this condition it may be impossible to differentiate the condition prior to operation.

7. *Mesenteric Lymphadenitis*.—The initial centralized abdominal pain and muscle spasm of acute appendicitis are absent, but otherwise the picture is remarkably similar. Most cases are diagnosed correctly only when the abdomen has been opened.

8. *Genitourinary Conditions*.—Hydronephrosis and pyelitis frequently mimic acute

appendicitis. The presence of blood or pus in the urine, the radiation of pain from the loin to the inguinal region, or genitalia, the palpation of a tender kidney, the demonstration of a renal or ureteric calculus by a skiagram are important. In pyelitis a catheterized specimen of urine will show gross pus, the fever is higher, and prostration may be present. Tenderness is present usually in the loin.

9. **Pneumococcal Peritonitis.**—This occurs in young girls in the lower socio-economic group between the ages of five and eight. It is usually primary, but occasionally may be secondary to pneumonia, or otitis media. When primary, the infection is thought to ascend by way of the patent Fallopian tubes. The symptoms are high fever, with diarrhea and vomiting. On palpation, a doughy, slightly distended abdomen is felt, in a thin young individual. The treatment is operative, to confirm the diagnosis and for the local administration of penicillin.

B. Colic, whether biliary or renal, is usually due to a stone passing down the duct and is usually severe. The patient moves about in agony, but is comfortable between attacks.

1 **Biliary Colic.**—The pain radiates to the back and shoulders, and there is usually a history of previous attacks. There is flatulence, indigestion, and occasionally jaundice.

2 **Renal Colic.**—There is frequency of micturition and the pain usually radiates from the loin to the genitalia. Urinary investigation revealing blood and pus cells in the urine, and the roentgenological demonstration of a calculus are important.

3. **Appendicular Colic.**—Appendicular colic is usually of an entirely different character from the other colics. It is better described as a sudden cramplike abdominal pain of varying intensity, with no relation to meals, and frequently accompanied by nausea, vomiting, and headache. Examination

is negative, unless the obstruction continues into obstructive appendicitis.

C. The presence of a mass in the right lower abdomen may be one of the following:

1. **Appendicular Abscess.**—This is one of the presenting signs of appendicitis.

2. **Ovarian Cyst.**—Follicular cysts are very common and frequently cause difficulty because they cannot be felt on examination. The pain caused by twisting of the pedicle of an ovarian cyst is made worse by movement and the size, usually that of a fetal head, is large enough to be felt on pelvic examination.

There are several other chronic conditions which may cause a mass or pain in the right lower abdomen, but the distinguishing features should be brought out by the history, examination, and special investigation; in a few cases the final diagnosis can be established only by laparotomy.

It should be stressed that acute appendicitis is the commonest surgical abdominal condition, and that most of the other conditions which have been mentioned are relatively uncommon. The most important differentiation is that of early pneumonia, when unnecessary operation is hazardous and must be avoided.

COMPLICATIONS

A. 1. Abscess is the commonest complication of appendicitis and, of necessity, means that the infection has spread through the wall or that the appendix has perforated and the infection has been localized by means of the omentum wrapping itself around the infected organ. The position of the abscess will depend on the position of the appendix. The four situations have already been referred to, but will be mentioned again.

(i). **Retrocecal** is associated with psoas spasm and is usually late in appearing. It may point in the groin.

(ii). **Pelvic.** When the appendix is in the pelvic position, the infection spreads down

aged. Many of the patients in these groups are left until perforation occurs because, until then, the signs are indefinite. In children, early pneumonia may simulate appendicitis, but the tenderness and spasm are less marked, the pain is frequently higher, the respiratory rate is increased and is frequently accompanied by movement of the abdominal wall and alac nasae. The fever, pulse rate, and leukocyte count are higher than in appendicitis.

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7. *Mesenteric Lymphadenitis*.—The initial centralized abdominal pain and muscle spasm of acute appendicitis are absent, but otherwise the picture is remarkably similar. Most cases are diagnosed correctly only when the abdomen has been opened.

8. *Genitourinary Conditions*.—Hydronephrosis and pyelitis frequently mimic acute

complications. Perforation may occur within a few hours and may be precipitated by the administration of cathartics. Only when operative removal is impossible is it permissible to temporize. The patient is placed in Fowler's position, nothing is given by mouth, and antibiotics are administered. Cold compresses should be applied to the abdomen to slow down the process and relieve the pain.

OPERATION

For the sake of clarity, the operative treatment will be divided into that for the ruptured and unruptured appendices.

Removal of the appendix is carried out under aseptic conditions; spinal or general anesthetic may be used.

The skin incision may be oblique, or transverse following Langer's line, with the center of the incision at McBurney's point. The subcutaneous fascia is next incised. The external oblique aponeurosis is opened in the direction of the fibers. The internal oblique and transversalis muscles are separated in the direction of their fibers; and by gentle traction, the peritoneum is exposed. This is carefully lifted and opened, and the cecum usually presents. If the finger is curled around the taenia, the cecum can usually be delivered through the wound. Where the three taeniae converge, the base of the appendix will be found. The appendix is then carefully delivered through the wound. The mesoappendix, containing the appendicular artery, is clamped, divided, and ligated. A purse-string suture is placed about the base of the appendix, and the surrounding area is covered with moist gauze. The base of the appendix is then clamped with forceps and the crushed portion tied near the base. The appendix is then severed with the actual cautery, and the stump of the appendix is invaginated by means of the purse string which is tied. The raw area of the mesoappendix is carefully covered. The small bowel should be examined for a distance of three

feet to rule out the presence of a Meckel's diverticulum and lymphatic glands in the mesentery of the small bowel. In females, the uterus and appendages are palpated with the two fingers. The wound is closed in layers with interrupted sutures, without drainage.

In some cases, this gridiron incision is not adequate, but when necessary may be extended medially to the sheath of the rectus muscle. When the appendix is adherent and cannot be delivered, it may be advisable to cut the base and dissect the appendix out in a retrograde fashion. When the diagnosis is in doubt, a lower paramedian incision rather than a gridiron should be used.

When a localized abscess is encountered and the appendix presents easily, the organ is removed as described above and the abscess is drained through the wound. If, however, an abscess is encountered and the appendix is not readily available, then it is wise merely to drain and leave the appendix to be removed at a later date, usually three months afterward. The danger of breaking down a natural barrier and spreading the infection is more serious than leaving the organ where the infection has already spread beyond its limits. If the cecum is edematous and friable, it may be impossible to invert the stump of the appendix, and in such cases the omentum may be gently sutured over the cecum as a further precaution.

Ruptured Appendix.—The perforated gangrenous appendix with a generalized peritonitis is a serious condition. The appendix is therefore removed as quickly and as gently as possible, and second, the peritoneal cavity should be drained. Usually the drains are placed in the most dependent position or where the infection is most likely to localize, such as the pelvis, retrocecal fossa or toward the ileocecal mesentery.

In localized abscesses, and, in fact, even in generalized peritonitis, topical application of penicillin and streptomycin into the cavity through a small catheter beside the drain has been of great value. The local concen-

into the pelvis and frequently causes irritation to the bladder and rectum, with frequency of micturition and diarrhea. On rectal examination there is a tender, boggy mass in the pelvis, which may be fluctuant.

(iii). When the appendix lies between the coils of the small intestine, the abscess is localized in between these coils and presents as an enteritis. A mass may be present, which, however, may be very difficult to feel because it is masked by the distended intestine.

the abdominal wall. This is soon followed by the Hippocratic facies and abdominal distention.

C. With peritonitis alone, the patient recovers, but should paralytic ileus develop, the prognosis is grave. It is due to severe toxic infection paralyzing the small bowel with increased distention.

D. Thrombosis of the portal vein is due to the infection spreading into the venous system. When this spreads to the liver, suppurative pylephlebitis develops, with chills

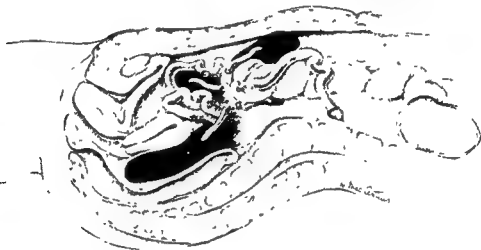


Fig 196—Sites of localized peritonitis depending upon position of appendix

(iv) The appendix lying next to the parietal peritoneum causes early irritation and is therefore usually dealt with before abscess formation takes place. However, the localizing adhesions or the omentum may form a mass in the peritoneal cavity near the anterior abdominal wall.

2. Subphrenic Abscess.—Subphrenic abscess is becoming less common after appendicitis and is usually a residuum of generalized peritonitis.

B. Generalized, spreading peritonitis with failure to localize the perforated or infected appendix is a serious complication. There is widespread abdominal tenderness, increased pulse rate and boardlike rigidity of

and high fever, and an enlarged, tender liver. Fortunately, with the use of the antibiotics and earlier operative interference, this complication is now rarely encountered.

E. Pulmonary embolism may occur after any operation, from thrombi originating in the veins of the legs and pelvis. Small ones make themselves evident by pain in the chest and spitting of blood. A large embolus usually occurs suddenly without warning about the tenth day and is commonly fatal.

TREATMENT

The treatment of acute appendicitis is the removal of the organ, as soon as the diagnosis is made. To delay only means to court

tration is about one thousand times higher than with parenteral administration, and the formation of adhesions has been minimal. This should be continued for 2 or 3 days using one-half million units of penicillin and one-half gram of streptomycin. Other antibiotics such as aureomycin, Chloromycetin, terramycin or neomycin may be added as desired.

EXPECTANT TREATMENT

The expectant treatment advised by Ochsner and Sherren is really the treatment of a localized abscess or inflammatory mass of some duration. It should never be used in the treatment of acute appendicitis except in cases occurring in isolated areas where adequate surgical facilities are not available. The patient is placed in the true Fowler's position at an angle of 30 degrees with the floor, hot fomentations are applied to the abdomen in order to assist in the resolution of the mass, nothing is given by mouth, and sedatives are given to relieve pain. If this procedure is followed the mortality is lower, and subsequent removal three months later may be carried out safely.

POSTOPERATIVE CARE

In the uncomplicated cases there is remarkably little postoperative treatment. Patients are encouraged to get up the next day and to lead as normal a life as possible.

In those cases with a localized abscess or generalized peritonitis, the patient is placed in Fowler's position, and gastric suction by means of a tube is used to prevent distention of the bowel and the development of adynamic ileus. Change of posture, complete abstinence of food and fluids by mouth and the intravenous administration of fluids and electrolytes also aid in accomplishing this result. Any attempt to stimulate the paralyzed bowel by drugs or surgery is futile.

Subacute Appendicitis

Subacute appendicitis may be a convenient term to describe those cases which are

milder than the acute form, and may include recurrent appendicitis, but does not include the specific granulomas such as tuberculosis and actinomycosis. There is no practical way of correlating these milder forms with etiological causes or pathological classifications except to say that they will include catarrhal inflammation, exudative, proliferative and productive inflammation, but exclude suppurative, ulcerative, and obstructive inflammation and necrosis of the appendix.

Recurring Appendicitis

The term chronic appendicitis, while in wide use in medical circles, does not fit in with any known pathological entity nor is there any clear-cut clinical picture. Vague symptoms of pain in the right lower quadrant, often ascribed to chronic appendicitis, are frequently not relieved by removal of the appendix.

However, the changes which occur in the appendix during an acute attack predispose to recurring infections. In recurring acute appendicitis the symptoms are similar to those described under the acute form although milder in degree, and between attacks the patient may be free of symptoms. If this clinical picture presents, the diagnosis is not in doubt and the patient will be cured by removal of the appendix.

More common is recurring appendicular colic. Fecaliths or other foreign bodies contained within the appendix cause excessive peristalsis in the organ as it attempts to expel the object. Colicky spasm felt in the right lower quadrant of the abdomen associated with deep tenderness on palpation in this area will confirm the diagnosis.

TUMORS OF THE APPENDIX

Benign

The general incidence of new growths of the appendix is low, consisting of about 0.5% of all intestinal growths; but the appendix may be frequently involved in sec-

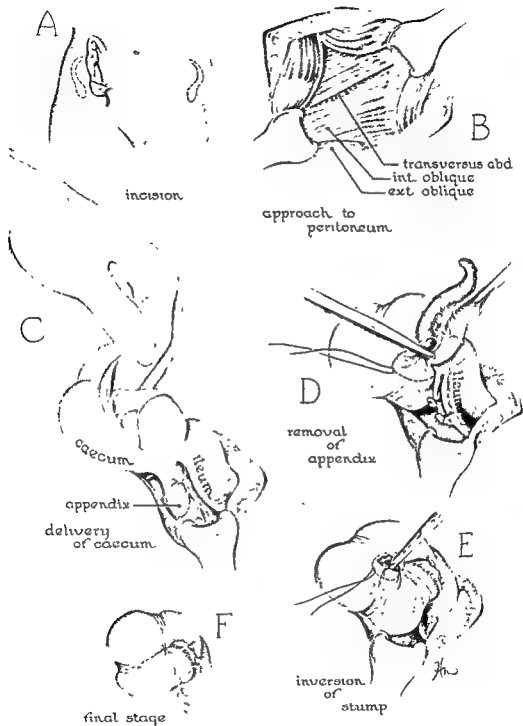


Fig 177

CHAPTER XXVII

COLON

H. S. MORTON, M.B.

EMBRYOLOGY OF THE COLON

The development of the gut begins at the sixth week of intrauterine life, when the alimentary tube grows faster than the body cavity, so that by the tenth week both the prearterial and postarterial part of the midgut are in the umbilical cord outside the peritoneal cavity. After this the peritoneal cavity grows faster than the midgut, which returns into the celom (body cavity) and rotates counter clockwise, the postarterial portion lying to the left. The cecum comes to lie in the right hypochondrium underneath the liver at the eleventh week, and then slowly descends to the right iliac fossa. Finally, the right and left portions of the colon, namely, the ascending and descending colon, lose their mesentery.

If this normal process is not completed, the various anomalies of malrotation occur.

- 1 Incomplete descent of the cecum.
- 2 Lack of attachment of the cecum, so that it is freely mobile.
- 3 True malrotation or clockwise replacement into the peritoneal cavity, with the small bowel in front of the large bowel

ANATOMY

The large intestine begins in the right iliac fossa as a blind pouch known as the cecum which extends about $2\frac{1}{2}$ inches below the ileocecal junction. It then continues as the ascending colon, the hepatic flexure, the transverse colon, the splenic flexure, the descending colon, the sigmoid, and finally ends at the peritoneal reflection in the pelvis.

The ascending and descending colon, and the two flexures are fixed posteriorly and

covered by the peritoneum, except on their posterior aspect. The transverse colon and the sigmoid, on the other hand, have mesenteries which permit wide mobility. The total length of the large bowel is usually 5 feet, approximately one-fifth that of the alimentary canal. The diameter gradually diminishes from about $2\frac{1}{2}$ inches in the cecum to $1\frac{1}{2}$ inches in the sigmoid.

The wall of the large intestine consists of five layers. There is a lining mucous membrane of single columnar cells which are arranged into the crypts of Lieberkühn. Under this is the submucosa containing the vessels, nerves, and lymphatics. Next comes the circular muscle coat. The longitudinal muscle fibers are characteristically collected into three bundles called the taeniae. These are about one-sixth shorter than the remainder of the intestine, and therefore the wall is puckered into large sacculations. The outermost layer is the serosa or visceral peritoneum covering the appendices epiploicae which consist of fat and blood vessels. The taeniae and appendices epiploicae are particularly characteristic of the large bowel.

Blood is supplied to the colon from two sources: the superior mesenteric artery gives off the middle colic artery and the right colic artery. This latter vessel divides into two branches: the descending branch anastomoses with the terminal branch of the superior mesenteric artery in the ileocecal region; the ascending branch anastomoses with the right branch of the middle colic artery. The left colic artery arises from the inferior mesenteric artery near its origin and passes upward and to the left where it divides into ascending and descending branches. The ascending branch anastomoses with the left

ondary neoplasm. The simple tumors, all of which are rare, include: fibroma, myxoma, lipoma, angioma and adenoma. This last is more common than the others, is usually associated with the symptoms of mild appendicitis, and is an accidental finding at operation.

Malignant

Primary malignant tumors of the appendix are uncommon. Adenocarcinoma occurs but is rare. Of more importance is the carcinoid tumor. These occur in the appendix and in the small bowel. The location of the tumors is of some importance, for if in the appendix they are of low malignancy and do not metastasize, while those in the small intestine may be multiple, produce obstruction to the bowel, and metastasize to the regional lymph nodes.

Carcinoid tumors of the appendix usually cause no symptoms and the diagnosis is made only at operation. They produce a firm localized swelling in the appendix and appear light yellow on their cut surface.

Microscopically the tumor consists of masses of spheroidal cells which are rich in lipid. According to Masson they are chromaffinomas.

The treatment is resection. Recurrence of carcinoid tumors of the appendix is rare and the prognosis is excellent.

REFERENCES

- British Surgical Practice: ed by Sir Ernest Rock
Carling and J. Paterson Ross, London, Butterworth, 1947-1950.
- Burton, J. A. G.: Appendix, Tumours of, p. 320.
Nuttall, A. G., Wardleworth: Appendicitis, Acute, p. 293.
- Cope, Zachary: The Early Diagnosis of the Acute Abdomen, ed. 2, New York, 1940, C.V. Mosby Co.
- Kell, J. H.: The Appendix, London, 1947, H. K. Lewis & Co., Ltd.
- Maingot, Rodney: Abdominal Operations, ed. 2, New York, 1948, Appleton-Century-Crofts Company, Inc.
- Wilkie, D. P. D.: Acute Appendicitis and Acute Appendicular Obstruction, Brit. M. J. 2: 959, 1914.
- Wilkie, D. P. D.: Observations on Mortality in Acute Appendicular Disease, Brit. M. J. 1: 253-255, 1931.

branch of the middle colic artery near the splenic flexure, while the descending branch anastomoses with the first sigmoid artery. All these branches give off short straight vessels to supply the colon. As they approach this organ, they divide into two branches which penetrate the muscle wall. Finer branches move around the surface to supply both the muscle and mucosa near the two anterior taeniae. The openings for these vessels through the circular muscle coat frequently cause weakness in this layer, particularly on the left side. The sigmoid arteries vary in number from one to six and anastomose with each other in arcades. There is a second series of smaller arches before the short straight arteries enter the sigmoid in the same manner as in the rest of the colon. The architecture of the vessels in the sigmoid is therefore similar to that of the small bowel.

The length of the sigmoid arcades is of fundamental importance in proctosigmoidectomy because it is only when the arcades are long that it is possible to mobilize the sigmoid down to the anus and preserve the blood supply.

The arterial architecture is accompanied by the venous tributaries, the lymphatic drainage, and sympathetic nerves. Many surgeons believe that the blood supply determines the extent of the surgery which can be performed, but it has been found in practice that end-to-end anastomosis can be executed in any part of the large bowel, and the blood supply is better than was previously believed to be the case.

The lymphatic supply begins in the submucosal layer with the vessels forming a network which drains into numerous glands along the posterior aspect of the colon and in the mesentery. These glands in turn drain by vessels to other glands in the middle of the mesentery and finally into the para-aortic glands. The limiting factor to cancer surgery is involvement of the para-aortic glands by the growth which makes them ad-

herent not only to the abdominal aorta but also to the inferior vena cava. Therefore surgery, to be successful, must be carried out before the disease has spread to these glands.

PHYSIOLOGY

One function of the colon is to concentrate the feces by removing water. The size of the colon varies with the diet of the individual animal. Meat-eating animals have very little need for a colon because digestion has been completed in the small bowel. It is therefore only necessary for the water to be absorbed from the feces. In herbivora, on the other hand, digestion of cellulose products takes place in the colon and there is usually a large cecal pouch where digestion may continue for a long time. Man's colon is designed for an omnivorous diet. It is slightly sacculated as in some of the herbivora, and the length is suitable for the digestion of a moderate amount of cellulose.

Movements of the Colon.—It is extremely difficult to observe peristaltic movement of the large intestine either at operation or during radiological studies. Occasionally a sudden movement may be observed on the fluoroscope, when the contents of the right half of the colon move over to the left half in a matter of seconds. This is called mass movement and usually occurs after a meal, in association with the gastrocolic reflex. There is a relaxation of tonic action of the musculature, the haustral markings disappear and the colon becomes a smooth tube. Then a large peristaltic wave sweeps the whole contents along.

The Nerve Plexuses.—The nerve plexuses are arranged in a somewhat similar manner to those in the stomach. In fact there appears to be a close association with the central nervous system of both the stomach and the large bowel. The autonomic nervous supply consists of the sympathetic and parasympathetic nerves; the sympathetic follow the blood vessels to the large bowel while the parasympathetic are the vagus, and the

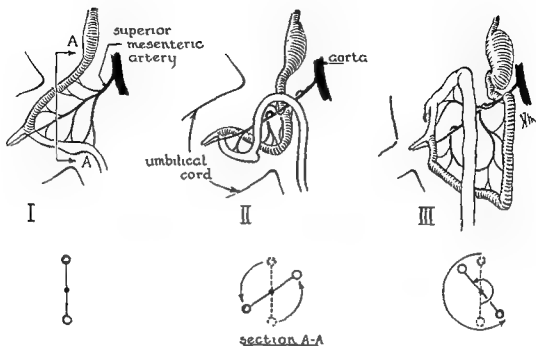


Fig 198—Rotation of the gut

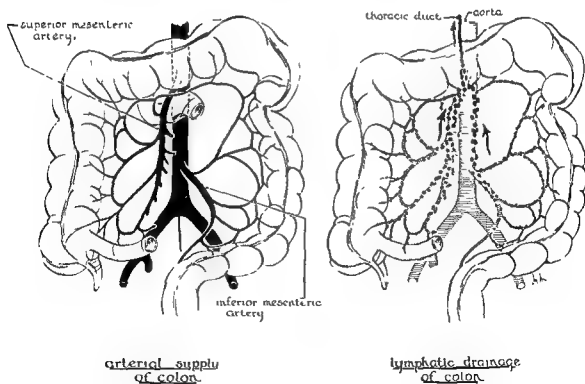


Fig 199.—Normal arterial supply and lymphatic drainage of colon

branch of the middle colic artery near the splenic flexure, while the descending branch anastomoses with the first sigmoid artery. All these branches give off short straight vessels to supply the colon. As they approach this organ, they divide into two branches which penetrate the muscle wall. Finer branches move around the surface to supply both the muscle and mucosa near the two anterior taeniae. The openings for these vessels through the circular muscle coat frequently cause weakness in this layer, particularly on the left side. The sigmoid arteries vary in number from one to six and anastomose with each other in arcades. There is a second series of smaller arches before the short straight arteries enter the sigmoid in the same manner as in the rest of the colon. The architecture of the vessels in the sigmoid is therefore similar to that of the small bowel.

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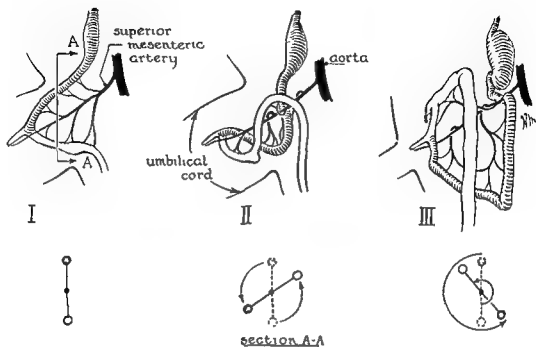
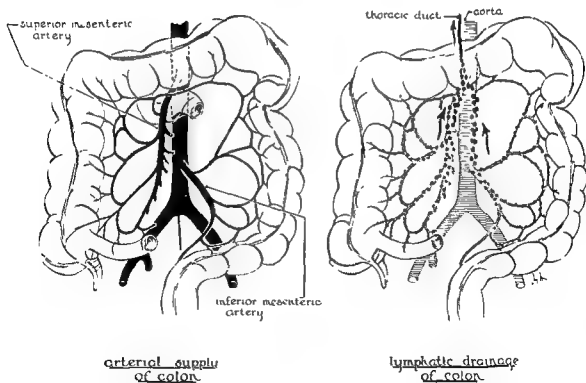


Fig 198 —Rotation of the gut.



arterial supply
of colon

lymphatic drainage
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sacral outflow from S2 and S3. As far as is known the vagus supplies the right half of the colon almost to the splenic flexure, and the sacral outflow the left half of the colon. The numerous plexuses in the colonic wall are still being debated, as there are more than those described by Auerbach and Meissner.

CONGENITAL ANOMALIES

Congenital anomalies of the colon are uncommon. They include congenital atresia, congenital stenosis, and duplication.

Congenital Atresia

Before the 5th week of intrauterine life the alimentary canal has developed into a tube with an epithelial lining. The epithelium then proliferates and fills the lumen. This process involves the whole alimentary canal. By the 12th week vacuoles appear and the lumen is re-established. Any arrest in this development during the 2nd or 3rd month may lead to atresia. This atresia may vary from a localized area which may be a thin diaphragm to complete involvement of the colon.

Clinical Picture.—Babies with atresia may have symptoms of vomiting from the first day of life, which is progressive and contains bile. Small amounts of modified meconium may be passed. The abdomen is distended, and dehydration rapidly ensues. A flat plate of the abdomen will demonstrate the dilated bowel. Farber's test may help in the diagnosis. In this test there is absence of the cornified cells normally present in the meconium.

Treatment.—Though the mortality is still high, only early operative treatment will save the life of the infant. At operation the distal bowel is distended with oil and air to increase its size as nearly as possible to that of the proximal bowel, and a side-to-side anastomosis is performed. If this is not feasible, a colostomy may be done.

Congenital Stenosis

Congenital stenosis, narrowing of the lumen, is embryologically a similar process to atresia. The lumen is always a single one, and only about half the cases show signs of obstruction, with vomiting, loss of weight, and constipation. The treatment is operative, with a primary side-to-side anastomosis. Dilatation should never be attempted, nor should it be necessary to perform a colostomy.

Duplication

In the development of the colon, two or more vacuoles may coalesce to form two lumina which may involve all or part of the large intestine. The wall of the duplicated bowel consists of all the layers of the intestine forming a second tube which lies in between the mesentery. At its distal end it communicates with the intestine. It is found most commonly in the midgut and less frequently at the upper and lower ends. It varies in size from a small blind pouch up to a second intestine. The principal symptoms are bleeding, associated with abdominal distention, and some variation in the bowel habit.

Duplication of the colon may present a normal appearance except for an extra taenia and a septum dividing the large bowel into two parts in a longitudinal direction. This may vary in extent from two complete colons and appendices to a septum in the rectosigmoid region of only a few inches in length.

Treatment will depend on the extent of the disease and the symptomatology. The septum in the rectosigmoid region may be crushed between clamps, while symptoms of bleeding or obstruction in one or other segment of the bowel may necessitate an ileostomy and total removal.

Enteric Cysts

Enteric cysts are localized duplications of the bowel. Single enteric cysts may occur anywhere between the tongue and the anus.

The wall consists of all the layers of the intestine and is attached to the intestine at one point. In the center is a clear mucoid fluid, usually opalescent, but it may be thick and opaque. These cysts do not cause symptoms until adult life, when there may be slight variation in the bowel habit, vague abdominal pain, and on examination, a tumor is palpable.

The differential diagnosis is from a mesenteric cyst which is lymphatic in origin, thin walled, and usually contains chyle. The treatment is removal, and this can usually be done without interfering with the blood supply to the bowel.

Congenital Megacolon (Achalasia of the Colon, Hirschsprung's Disease)

Congenital megacolon, usually occurring in infants, is characterized by obstinate constipation and marked distention of the colon. Hirschsprung first described the disease in 1868, believing that the hypertrophied and dilated colon was the primary site of the lesion, and his views went unchallenged for about sixty years. It was not until 1945, when Cassey called attention to a persistent narrowing of the lower rectal and sigmoidal segments, that Swenson and Hiatt reinvestigated the whole problem. By means of balloons inserted through a colostomy, Swenson proved that a normal peristaltic wave did not pass through the rectum. They also investigated the distal segment and found the autonomic ganglion cells of Auerbach to be absent or greatly reduced, so that there is a loss of the fundamental gut reflex of progressive peristalsis. They were also struck by the fact that few cases are seen in adults and by the more frequent occurrence in children. It was soon apparent that congenital megacolon was a dangerous disease and that many children died from perforation, ulceration, and from a state of shock resulting from frequent use of enemas of tap water which is hypotonic and rapidly absorbed from the huge bowel

Symptoms.—Obstinate constipation is the usual complaint, which is sometimes noticed soon after birth. Days or even weeks may elapse without a bowel movement. Symptoms of partial obstruction may occur, with occasional bouts of diarrhea, which is really an overflow. Laxatives and enemas have little effect. The dilatation of the colon produces a distention of the abdominal wall; but subjective symptoms are usually not pronounced. Occasional abdominal discomfort occurs with perhaps some dyspnea. As the disease progresses, constitutional symptoms appear, similar to those of intestinal obstruction. Death may be caused by pneumonia, secondary infections or complete obstruction, as well as from the acute complications mentioned above.

Diagnosis.—Functional constipation, anal stricture, and even malignancy must be excluded. A barium enema is essential. The rectosigmoid narrowing is best visualized as the barium is run in. Care must be used to remove as much as possible of the barium so that impaction will not occur.

Treatment.—The older operations of colectomy, ileosigmoidostomy and sympathectomy have given place to resection of the obstructing segment, with preservation of the anus. The operation of Swenson, or the modification of Hiatt, has yielded excellent results in a large percentage of cases. Essentially it consists of resecting the lower sigmoid and rectum, and restoring the continuity of the bowel. A temporary colostomy may be necessary to improve the patient's nutrition and prepare the bowel for a safe anastomotic procedure.

FOREIGN BODIES

Swallowed foreign bodies commonly pass through the alimentary canal; occasionally they may become lodged in the colon, usually the sigmoid; they may also be introduced from below or travel from an adjacent structure. Several factors contribute to the lodgment of foreign bodies in the sigmoid colon

since its lumen is narrower and there is a physiological sphincter at the rectosigmoid junction. Those introduced from below seldom get beyond the rectum. Penetrating foreign bodies are rare but occasionally impaling accidents may cause them to break off and remain in the lumen.

The symptoms are extremely variable. There may be pain, hemorrhage, constipation or diarrhea, or the complications of infection or genitourinary tract involvement.

Treatment.—Unless peritonitis has occurred, the treatment is expectant as most foreign bodies will pass spontaneously. If peritonitis results from perforation of the bowel, laparotomy must be performed. Rectal perforation may result in a pelvic cellulitis which should be drained.

VOLVULUS

Volvulus is a torsion of the bowel on its axis, usually resulting in obstruction. Most cases occur in the sigmoid region because of the length of its mesentery, although it may occur in the cecum and terminal ileum.

Clinical Picture.—The picture is one of acute severe intestinal obstruction. The abdominal distention is marked, and copious vomiting occurs. There is early dehydration and shock, particularly if strangulation of the involved loop has developed. There is frequently a history of similar but less severe attacks of pain from which the patient has recovered.

A flat film of the abdomen will show greatly dilated loops of bowel, and it is frequently possible to ascertain the area of volvulus by the position of the dilated loops.

Treatment.—Treatment is laparotomy and untwisting of the involved loops of bowel. If strangulation has occurred, resection is necessary. The accompanying shock and dehydration must be corrected by intravenous fluids, electrolytes and whole blood transfusion. Following untwisting, the volvulus may recur.

DISEASES OF THE APPENDICES EPIPLOICAE

The appendices epiploicae are localized, peritoneal covered out-growths of fat from the wall of the large bowel, and are one of the main characteristics of the colon. Their number varies enormously and may be in excess of two hundred. They are most numerous in those areas with a mesentery, namely, the transverse and sigmoid colon, and are arranged in two parallel rows adjacent to the anterior and posterior longitudinal taeniae. One of these appendices may become twisted, causing a sudden sharp abdominal pain which is referred to the right side of the abdomen. Vomiting, tenderness, and rigidity are present, giving the picture of an acute surgical condition which may be mistaken for appendicitis, cholecystitis, or diverticulitis. The treatment is surgical removal. The appendices epiploicae may become adherent to other abdominal contents and cause intestinal obstruction. This is usually a late result of infection. No definite symptomatology can be given.

DIVERTICULOSIS

True and false diverticula occur in the large bowel, the former are rare, amounting to less than 1%, and occur in the proximal half of the colon. Their walls consist of all the layers of the bowel. False diverticula generally occur in the distal portion, are nearly always multiple, and consist of a herniation of the mucosa through the musculature, usually where the vessels penetrate the wall. They are caused by increased intraluminal pressure. They are frequently seen in barium studies of the colon and do not cause any symptoms.

Pathology.—Diverticula are found in 5% of persons over 40 years of age. If the opening of the sac into the intestine is large, symptoms will rarely develop, as the sacs empty. However, when the openings are small, the contents of the bowel enter the





false diverticula and may become lodged there, leading to ulceration of the walls and the production of diverticulitis.

DIVERTICULITIS

Inflammation in a false diverticulum is diverticulitis. The signs and symptoms are those of localized intra-abdominal inflammation, pain, tenderness and rigidity, usually in the left lower quadrant of the abdomen. Perforation of a diverticulum may result in a localized abscess or generalized peritonitis.

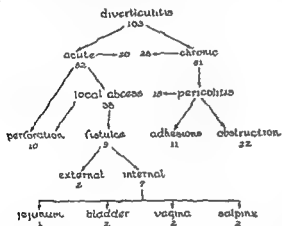


Fig 200 —Table of 103 cases of diverticulitis with complications.

If an abscess develops, a tender mass will be palpable. The abscess may rupture, causing generalized peritonitis, or it may communicate with adjacent bowel or bladder, resulting in a fistula. These fistulas may be internal, or external onto the skin of the abdomen or perineal region. Because of the age of the patients, it may be difficult to rule out malignancy and careful investigation must be carried out. Occasionally the inflammatory process may obstruct the lumen of the bowel.

Treatment.—Unless obstruction, abscess formation, perforation or fistula formation occurs, the treatment is conservative.

ULCERATIVE COLITIS

Etiology.—By definition the cause of non-specific ulcerative colitis is unknown. Vari-

ous theories have been advanced. Barger of the Mayo Clinic has supported an infective etiology. Others believe it to be an allergic phenomenon but this has only been confirmed in about 10% or less of the cases. The psychiatrists state that the patients are usually psychologically dependent on one member of the family group, that they are frustrated and not ambitious. These mental conflicts are transmitted through the vagus nerve to the colon, where it has been demonstrated that the impulses affect the blood supply causing first ischemia, later followed by a marked congestion. On the other hand, most authorities are still in doubt whether this psychological theory is a cause or an effect. Any of the normal flora of the colon may penetrate the mucosa and initiate minute inflammatory foci. Some have suggested a vitamin deficiency, and others neurovascular imbalance.

To simplify this complicated picture, it would seem wise to combine these theories into a composite picture. Persons with a certain psychological pattern may send impulses down the parasympathetic to the colon thereby altering the blood supply and allowing the normal bacterial population better access to the mucosa. The congestive phase favors aerobic organisms, the ischemic phase anaerobic, so that infection is a secondary manifestation in an already abnormal bowel. With the presence of ulceration there is the loss of absorption, vitamin deficiency, and alteration of nutrition, with resulting further diarrhea and the setting up of a vicious cycle.

Clinical Picture.—Nonspecific ulcerative colitis usually presents between the ages of 20 and 40 years but may occur at any age, and the sexes are almost identically affected.

In the true form of recurrent ulcerative colitis the attacks begin with severe diarrhea with frequent stools up to 15 to 20 a day, lasting more than a week. The diarrhea is associated with pain, malaise, weakness, loss of appetite, and loss of weight. The patient is pale, the eyes sunken, the ab-

domen slightly tender with no palpable masses, and the temperature is elevated. On sigmoidoscopic examination the colonic mucosa is red, granular, and bleeds easily to the touch, and pinpoint ulcers may be found. These pinpoint ulcers gradually enlarge until the muscle coat is exposed. Between the ulcers the remaining mucosa is engorged and edematous, forming pseudopolyps which occasionally may become malignant. The ulcers have even been noted to arrange themselves in three longitudinal bands in extensive cases. Radiological examination of the large bowel by barium enema shows an irritated, hyperactive colon with loss of normal haustration. In the more advanced cases, it has a "pipe-stem" appearance and ulcers are seen. Laboratory tests reveal low plasma proteins, and in severe cases, the liver function is impaired.

Complications.—The *minor* complications are hemorrhoids, fissures, pruritus, hypertrophic osteoarthropathy and infantilism. The *major* complications are pseudopolyposis, stricture, perirectal abscess, fistula, perforation, and carcinoma.

Differential Diagnosis.—The diagnosis of this disease is one of exclusion. At the time of sigmoidoscopic examination a swab culture is taken from the mucosa in order to exclude *Entamoeba histolytica* and the organisms of Shiga and Flexner which cause bacillary dysentery. Sigmoidoscopic examination will be negative for the 10% of the cases in which the disease is confined to the proximal colon, but this will be demonstrated by a barium enema. This examination will also determine the extent and stage of the disease. Malignant changes can only be excluded by biopsy, and supervene in about 5% of cases.

Treatment.—Medical treatment consists of blood transfusions and iron therapy for anemia, increased fluid intake to overcome dehydration, supplementary feedings of high protein milkshakes, and vitamins, particularly B and C. Intestinal sedatives should

be given, such as atropine, hyoscine and phenobarbital. This therapy is successful in more than half of the cases. A further 20% will be continued on medical treatment but with definite impairment of function. About one-quarter of the cases will require surgical treatment.

Indications for Surgery.—The surgical therapy of ulcerative colitis should be restricted to only the most carefully selected cases. For this purpose ulcerative colitis is divided into three groups:

Group I. Those cases with fairly mild symptoms that respond well to medical therapy and where the disease does not prevent regular attendance at work, or interfere with the daily routine of life.

Group II. Those cases of moderate severity with only fair response to medical treatment, frequent exacerbations of symptoms and a reduction of activity. This group is also subject to the complications or sequelae of this disease which are:

- 1 Perforation of the distended colon
- 2 Stricture formation.
3. Massive hemorrhage
- 4 Fistula formation
- 5 Abscess formation
- 6 Chronic inanition.
- 7 Psychological changes
- 8 Pseudopolyposis
- 9 Malignant change.

Group III. The fulminating type. These patients develop an extremely severe type of the disease with almost continuous diarrhea, hemorrhage, abdominal pain, dehydration, and rapid wasting.

Operative treatment of ulcerative colitis should be reserved for patients in Groups II and III. Group III patients should be prepared for operation by concerted efforts to improve the nutritional status, restore the blood volume, and reduce the pain and secondary infection. It may be necessary to operate on a woefully ill patient, but this is frequently the only means of saving life.

Group II patients, of course, require surgery for the complications of the disease. However, some Group II cases without complications require operative treatment, although great care in selection must be taken. We consider that operation is indicated under the following conditions:

- (a) If there is loss of time from work.
- (b) If pleasures or social activities are affected.
- (c) If pathological changes develop.

In any case, an ileostomy should not be performed until it becomes a most welcome relief from the tortures of the disease. It is our experience that if the ileostomy is done early in these so-called intractable cases the patients do not become psychologically adjusted to the ileostomy and are a continual problem for the surgeon. On the other hand, when the escape from a hitherto intolerable situation is given by ileostomy (and colectomy) the relief of the patient is such that he never wishes to go back to his old way of life.

Operative Treatment.—The operative treatment of ulcerative colitis has been unsuccessful in the past. Many different staged procedures have been recommended. It would now appear unnecessary except from a historical point of view to mention them—appendicostomy, cecostomy, and internal defunctioning anastomosis.

1. *Ileostomy.*—This alone carries with it a mortality of between 15 and 20%. It does not remove the disease and has been recommended in the past as a primary procedure to be followed in convenient stages by a right, then a left hemicolectomy, and finally abdominoperineal resection.

2. *Partial or Subtotal Colectomy.*—When medical treatment fails, the patients with fever, dehydration, anemia, continuing diarrhea, and blood, pus and mucus in the stool, should be prepared for operation by intensive intravenous therapy including blood transfusions, supplementary feedings by mouth, sulfonamides, antibiotics and intesti-

nal sedatives. The hemoglobin should be at least 80% and the plasma proteins within normal limits. Then an ileostomy combined with a subtotal colectomy should be carried out in one stage. This procedure eliminates the toxic absorption from the diseased bowel and the protein and the red cell loss in the bowel exudate. The mortality from this procedure is much less than from ileostomy alone even in fulminating cases, but it is important that this surgical procedure should be carried out early enough to be lifesaving. Some surgeons have attempted, in the very severe cases, to do a total colectomy with abdominoperineal resection in one stage. This, however, carries a high mortality and would seem to be too extensive.

A small proportion of cases, where the disease is limited to the proximal colon, may have a subtotal colectomy and an ileosigmoid anastomosis. Otherwise, the patients have to carry a permanent terminal ileostomy. The question is often raised whether it is possible to close the ileostomy and join it to the remaining sigmoid, rectum, or anus. It is very seldom that this is possible because the remaining bowel, although put to rest, does not heal.

3. In early cases, *vagotomy* has been advocated, particularly if the disease is limited to the proximal colon.

4. *Abdominoperineal resection* should be carried out if the disease process persists in the distal remaining segment.

Prognosis.—The patient with an ileostomy and a total colectomy can resume normal activity. There is no difficulty in maintaining satisfactory nutrition on a normal diet. The mortality for this procedure is less than 5%.

INTUSSUSCEPTION

Intussusception is an important and common cause of acute abdominal emergencies in childhood. By definition it is an invagination of a proximal portion of the bowel (the *intussusceptum*) into a distal portion (the

intussusciens). Asymptomatic intussusception is fairly common as has been established by observation at laparotomy for various causes on young children. This physiological intussusception is usually found in the small intestine and presumably reduces itself spontaneously.



Fig 201—Intussusception of the small bowel into the large (ileocolic)

Etiology.—The etiology of this condition is unknown, except for those cases which occur at the site of an intestinal polyp or Meckel's diverticulum. It seems possible that if an area of physiological intussusception occurs at the site of such a lesion, the invagination may not be reduced, and so progresses to the full-blown clinical condition. That such mild invagination can occur at the ileocecal region, the commonest site of organic intussusception, is quite possible, and anatomically feasible. It is put forward therefore as a likely etiological factor in this condition.

Pathology.—Intussusception may occur in almost any area in the small or large bowel, and may be single or more complex, in that

a second invagination follows the first or primary one. Occasionally the condition may be multiple and occurs in more than one area.

The following types of intussusception have been described:

1. Ileocolic.
2. Jejunocolic.
3. Ileocecal
4. Ileocolocolic.
5. Colocolic.

Once the intussusception becomes established, edema of the bowel sets in, there is peritoneal exudate, blood vessel strangulation, and finally gangrene if the condition goes unrelieved.

Clinical Picture.—Intussusception occurs most commonly in the first year of life and more often in boys than girls. The infant is usually well nourished and healthy in every way.

The cardinal symptom is severe, colicky abdominal pain, of spasmodic character. The child usually screams during the bouts of pain and may show signs of shock. Vomiting frequently accompanies the pain. The child may appear quite normal in the intervals between attacks of colic.

The classically reported signs of dehydration, severe shock, and bloody stools are later manifestations, and the diagnosis should be made prior to their development.

Abdominal examination will reveal the presence of a mass in almost every case. The presence of a sausage-shaped intra-abdominal mass with the history of severe crampy abdominal pain is pathognomonic of intussusception.

Rectal examination may reveal a tender abdomino-pelvic mass, a presenting intussusception and blood on the examining finger.

Aids to Diagnosis.—Examination of the abdomen under anesthesia should be done if no mass can be palpated in a suspected case.

Barium enema will reveal the intussusception in the ileocolic, or colocolic variety. Barium by mouth is never indicated.

Treatment.—Occasionally a water or barium enema will reduce an intussusception. Of necessity this method is only of value if the colon is involved in the intussusception. This procedure should be restricted to the very early case, or if surgical facilities are not available.

The treatment of choice is laparotomy and reduction of the intussusception by taxis. If this is carefully tried without success, the affected area of bowel must be resected and a primary anastomosis of the two ends of bowel carried out. As resection in a very ill child can be hazardous, a safer procedure is to exteriorize the intussusception. This can later be resected when the initial shock of the condition is over, and finally at a third stage the anastomosis can be performed.

Extreme care must be taken to replenish fluid and electrolyte deficits and to maintain the blood volume before and during the operative procedure. The gastrointestinal tract must be kept deflated by continuous nasogastric suction.

Occasionally intussusception will recur either at the same site or at another. The treatment does not differ in any way from that of the first attack. However, a second occurrence is usually diagnosed very early so that resection of injured bowel is rarely necessary.

Prognosis.—The mortality from this condition is now fortunately low—even when a laparotomy and bowel resection become necessary; as the knowledge of supportive therapy in children increases it will be even lower. The earlier the diagnosis is made and treatment instituted, the less the danger from the intussusception.

SPECIFIC INFECTIONS OF THE COLON

Tuberculosis

Tuberculosis is becoming steadily less common. When it occurs it affects the ileum, and is due to swallowed tubercle bacilli in a patient with pulmonary tubercu-

losis. In the region of the cecum it usually causes a large inflammatory mass which is palpable. It can be confused pathologically and clinically with terminal ileitis.

The treatment is a short circuiting operation or resection and subsequent anastomosis.

Actinomycosis

Abdominal actinomycosis commonly affects the cecum, terminal ileum, and appendiceal regions. It cannot be recognized preoperatively unless there are sinuses when the ray fungus can be demonstrated in the discharge.

The treatment is surgical removal and chemotherapy. (See Bacteriology.)

BENIGN NEW GROWTHS

All varieties of benign growths may be found in the colon. The most common of these are lipomas and adenomas. They may give rise to bleeding, diarrhea, and occasionally pain.

FAMILIAL POLYPOSIS OF THE COLON

Familial polyposis is a relatively rare condition characterized by the appearance of multiple adenomatous polyps in the colon and rectum. It is termed familial because it is transmitted from one generation to the next usually as a Mendelian dominant although occasionally as a recessive character. Although polyps may be present at birth, they usually appear later and produce symptoms between the ages of 15 and 40 years. Rarely the condition exists throughout life without causing symptoms.

Diagnosis.—Except when the family history of the condition is known and an apparently well member of the affected family is examined specifically for the presence of polyps, the diagnosis is made after symptoms of complications develop. Common symptoms are: (a) bleeding by rectum, (b) intussusception, (c) protrusion of a low-lying polyp from the anus, (d) bowel ob-

struction, chronic or acute, as a rule due to a carcinoma developing in a polyp

Almost invariably the diagnosis can be made by rectal and sigmoidoscopic examinations. The barium enema x-ray examination, particularly with air-contrast technique, will reveal the presence of the polyps in the bowel above the reach of the sigmoidoscope, provided they have reached sufficient size. When malignant degeneration of a polyp has occurred, the findings will be those of carcinoma of the colon or rectum.

Treatment.—Because each of the multiple polyps bears malignant potentialities, almost every individual with familial polyposis will sooner or later develop carcinoma of the bowel. For this reason prophylactic excision of abnormal mucosa is necessary.

There are two principal methods of treatment of multiple polyposis before malignant degeneration has occurred:

(a) Excision of the cecum, appendix, colon and rectum, with the establishment of a permanent ileostomy.

(b) Excision of the colon and upper portion of the rectum, with anastomosis of the ileum to the remainder of the rectum. After the latter operation benign lesions in the remainder of the rectum are removed by fulguration through a proctoscope.

The rectum must be removed when the rectal polyps are very large and too numerous for safe removal by fulguration.

MALIGNANT TUMORS OF THE COLON

Carcinoma of the colon is apparently becoming more frequent because of two factors: the increasing accuracy of diagnosis, and the lengthening span of life. During the twentieth century, this has increased by between 15 and 20 years, so that a larger number of people are entering the cancer age.

Carcinoma of the large bowel is a common disease, accounting for 10% of all cases of cancer.

The site of the growth in the large bowel depends on the accurate definition of the rectosigmoid. In order to overcome individual interpretation of this area, it is better to consider the colon and rectum as one unit. The following table shows the proportional involvement by site in this hospital.

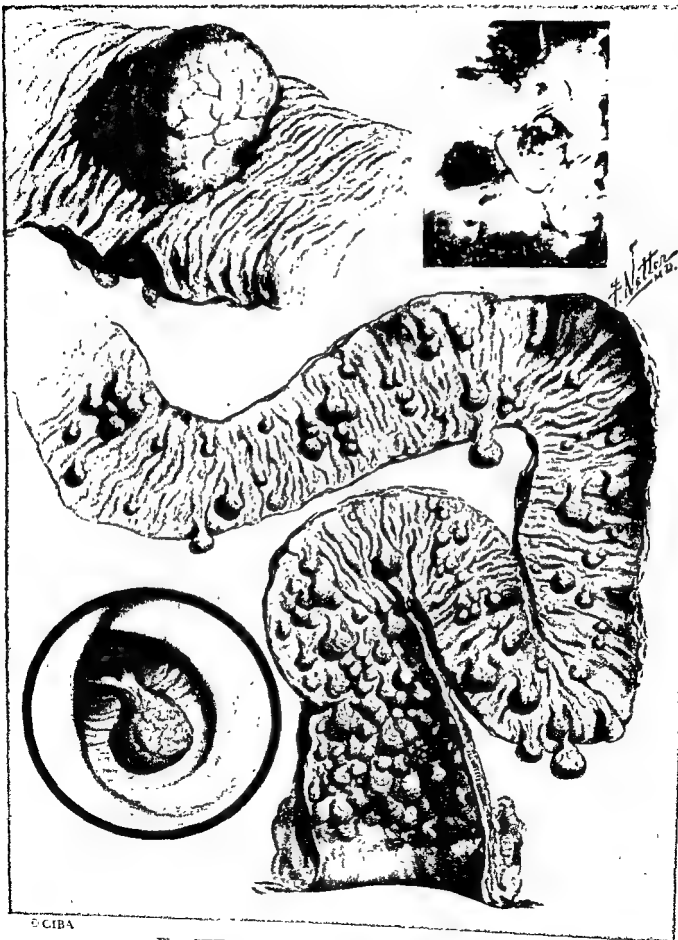
DISTRIBUTION BY SITE

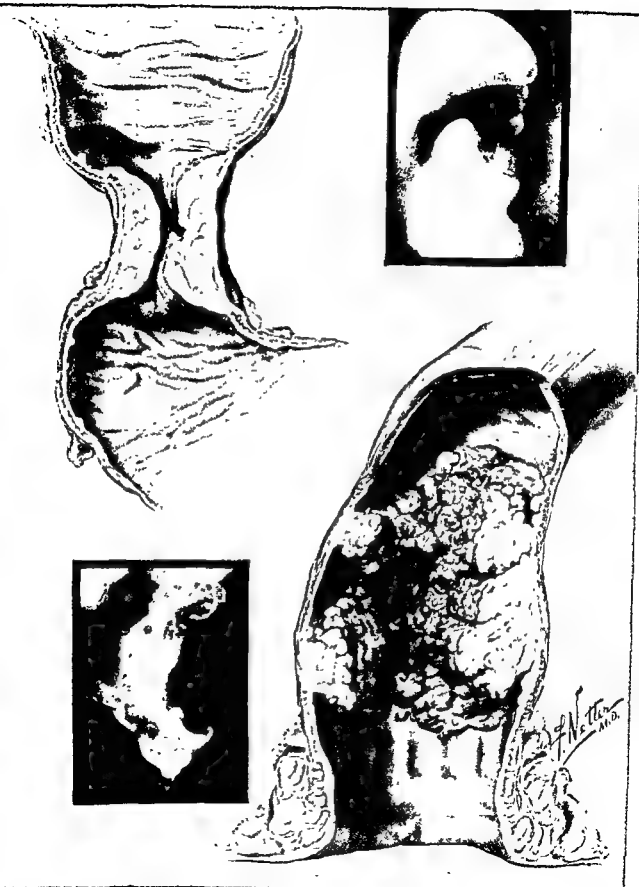
| | |
|-----------------|-----|
| Cecum | 7% |
| Ascending Colon | 10% |
| Transverse | 14% |
| Descending | 5% |
| Sigmoid | 21% |
| Rectosigmoid | 7% |
| Rectum | 36% |

Etiology.—The origin of cancer of the colon is not known but there are several predisposing conditions to cancer in this site. Of these, the most important is familial multiple polyposis which invariably becomes malignant. Single isolated polyps may occasionally undergo malignant change while pseudopolyposis becomes cancerous in approximately 2 to 3% of cases. Diverticulitis and cancer of the colon co-exist in from 3 to 5% of cases.

Pathology.—There are two main types of carcinoma in the colon. The commoner is the annular stenosing obstructive growth which is more frequent on the left side of the bowel. The proliferative cauliflower-like papillary variety occurs characteristically on the right half of the colon. They are both adenocarcinomas. Colloid or mucoid degeneration occurs in about 5% of cases.

Benign polyps may have a malignant change at their tip which can be called a carcinoma-in-situ. Invasive carcinoma arising in the polyp will show changes in the ground substance, but the growth may still remain outside the lamina propria. If the growth occurs at the base rather than at the tip of the polyp, it is more dangerous from





two points of view: it is nearer to the lamina propria and second, local removal is liable to failure because the polyp may be removed and yet leave a portion of the malignant growth. The next boundary line is the muscularis mucosa, then the layer of circular musculature. Malignant cells may be picked up in either the lymphatics or blood vessels from the lamina propria onward. The grading of pathological types by Broder's classification or Dukes' stages is of some value. No staging can be accurately done without pathological assistance. The present convention of staging as applied to the rectum could be conveniently applied to the colon:

Stage A.—Confined to the mucosa

- 1 Carcinoma-in-situ.
- 2 Carcinoma invasive.

Stage B.—Involving circular musculature.

Stage C₁.—Local lymphatic glands involved but removed surgically.

C₂.—More distant lymphatic glands involved possibly surgically removable.

Stage D.—Generalized dissemination of malignant disease

The spread of carcinoma of the colon is usually by the lymphatics but the other two methods should not be forgotten; namely, direct spread, and by means of the blood stream carrying the cells to the liver. This latter method is not quite as common in the colon as in the rectum. It is earlier in the flat sessile growths than in the papillary type. Metastases may also be found in the peritoneum and in the lung.

Clinical Signs and Symptoms.—Carcinoma of the colon is a relatively silent disease, and the early symptoms are vague and indefinite. Consequently it is frequently diagnosed only in the late stages. Vague complaints, such as weight loss and fatigue, must be followed up thoroughly. There is usually some type of dyspepsia, constipation, or mild alteration in the bowel habit, such as a frequency in the desire to defecate. Diarrhea, either spurious or blood-stained, is always

worthy of careful investigation, as is anemia, especially if it does not yield readily to medical treatment. Pain is usually late, but may be the presenting symptom. When cramplike in nature, it is evidence of early obstruction; when spastic, or associated with tenesmus, there may be a growth in the distal sigmoid or rectum. Steady pain in the right iliac fossa may be due to distention of the cecum, with a competent ileo-cecal valve and distal obstruction. A tumor may be felt by the patient or discovered accidentally on routine examination. Any mass without pain in the course of the large intestine must be regarded with grave suspicion.

Right Half of the Colon.—In this segment of the bowel, large cauliflower-like growths are common. These interfere with the normal function of absorption and give rise to anemia, intoxication, or a mass, which may be felt on examination. There may be a mild alteration in bowel habit, and bleeding may occur which should stimulate further investigation.

Left Half of the Colon.—Carcinomas in this portion cause the obstructive syndrome. The scirrhus growth spreads in the wall causing a gradual narrowing of the lumen with the onset of acute or chronic obstruction. This is characterized by visible peristalsis, borborygmi, distention of the abdomen and pain in the right iliac fossa due to enlargement of the cecum when the ileo-cecal valve is competent. Progressive constipation is the rule, while blood in the stool, although less common, is of more dramatic significance.

The stools may be small in caliber, or contain blood, either macroscopically or microscopically. Pus in association with blood suggests a carcinoma. This vague pattern of many signs and symptoms should be thoroughly investigated in order to establish, or disprove, the presence of malignancy.

Diagnosis.—The diagnosis is made by a careful history. Any alteration in the bowel habits of a patient of the older age group im-



ileo-transverse
anastomosis with
R hemi-colectomy



transverse-sigmoid
anastomosis
with L hemi-colectomy



abdomino-perineal
resection with
terminal colostomy



resection of
transverse colon



resection of
splenic flexure



resection of
sigmoid colon

Fig 202 —Types of operation for carcinoma of the colon

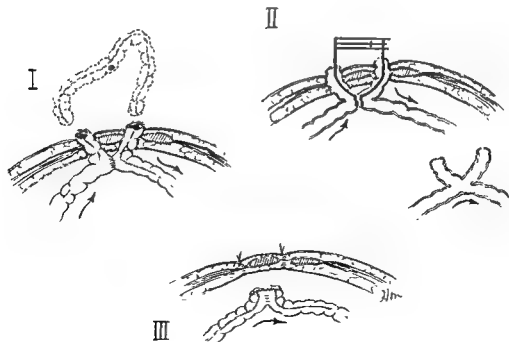


Fig 203 —Modified Devine's defunctioning colostomy and Mikulicz resection

mediately indicates a clinical examination of the abdomen for the recognition of any palpable masses and the noting of any abdominal distention.

This should be followed by digital and sigmoidoscopic examination although most colonic growths are above the reach of the examining finger and even the sigmoidoscope. Therefore an accurate diagnosis requires a barium enema.

Treatment.—The treatment is entirely surgical. Radiation therapy has little effect, so the only hope is early and complete removal. The preoperative preparation of cases with carcinoma of the colon has been gradually improved due to control of anemia by blood transfusions, of protein metabolism by concentrated feedings, both by mouth and intravenously, relief of distention by Miller-Abbot tube, and the sterilization of the bowel by chemotherapeutic agents. The nonabsorbable sulfonamides are given for 5 or 6 days before operation and 3 days before operation, one of the antibiotics affecting the gram-negative organisms is added. Streptomycin 2 Gm. a day, aureomycin 1 Gm. daily, Chloromycetin 4 Gm. daily, terramycin 3 Gm. daily, may be used. These are all given by mouth in powder, capsule or tablet form.

The synergistic action of the sulfonamides and antibiotics will control the flora and al-

low healing of the anastomosis. The bacterial content of the feces both aerobic and anaerobic can be considerably reduced, the only caution being that this is limited by a time factor to a 10-day period.

The cases may be classified into operable and nonoperable, obstructed and nonobstructed. In the nonobstructed operable cases on the right side, a right hemicolectomy with ileotransverse anastomosis should be performed. In the transverse colon, splenic flexure, descending colon and sigmoid, resection with end-to-end anastomosis is indicated; in the rectosigmoid and rectum, an abdominoperineal resection is the usual procedure when end-to-end anastomosis is impracticable.

In the obstructed cases, relief of the obstruction by proximal colostomy or cecostomy may be followed later by an appropriate resection and anastomosis; in the obstructed and nonoperable cases, the palliative colostomy or cecostomy will relieve symptoms; while in the last group of inoperable cases without obstruction, palliative supportive treatment alone is indicated. Postoperatively local and systemic antibiotics may be given.

REFERENCES

For references for this chapter, see those at the end of Chapter XXVIII.

CHAPTER XXVIII

RECTUM AND ANUS

H. S. MORTON, M.B.

INTRODUCTION

The incidence of disease in the rectum and anus is high. It occurs in all walks of life, at all ages, and in both sexes, and it is responsible for considerable loss of time.

ANATOMY

When palpating the anal canal, there are three important landmarks: the anorectal ring, the intramuscular depression, and the subcutaneous external sphincter. An understanding of these structures and their functional importance is essential for both diagnosis and treatment.

The *anorectal ring* consists of four parts: internal sphincter, longitudinal muscle, puborectalis, and deep external sphincter.

The *internal sphincter* is surrounded by the longitudinal muscle and the puborectalis, a specialized portion of the levator ani. The latter passes from the posterior surface of the pubic bone backward around the rectum, making a sling at the anorectal junction, causing the right-angled bend of the rectum with the anus.

The *external sphincter* is divided into deep, superficial, and subcutaneous portions. The deep and superficial portions are separated by a thin septum, while there is a well-marked fibromuscular septum between superficial and subcutaneous sphincters.

The lining epithelium of the rectum consists of columnar mucosa, which gradually becomes flattened and cuboidal, and gath-

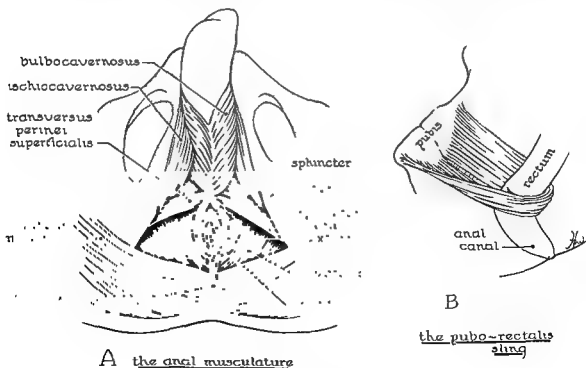


Fig. 204.—The anal musculature.

ered into longitudinal folds named the columns of Morgagni. There are seven to ten in number and are joined together below by the anal valves. At the apex of these valves, the mucosa becomes transitional, and quickly changes into stratified squamous epi-

thelium and two on the right of the anal canal which drain into the superior hemorrhoidal veins. The left lateral plexus is in the 3 o'clock position, the right posterior in the 7 o'clock and the right anterior in the 11 o'clock positions. It should be understood that the

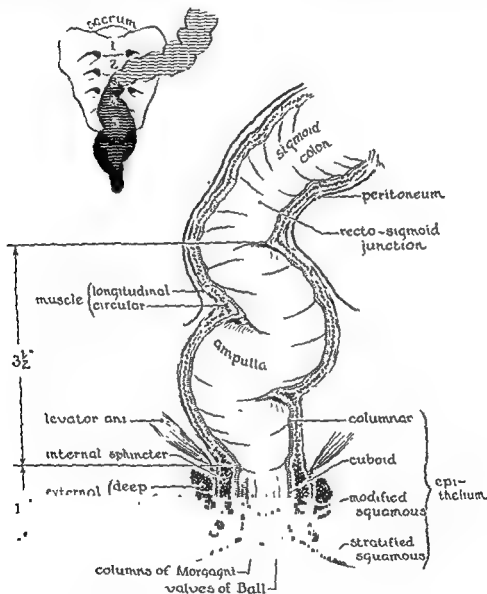


Fig. 205—Diagrammatic anatomy of the rectum

thelium, and true epidermis. Behind the anal valves there are crypts which occasionally collect fecal matter, and are also the site of vestigial anal glands, which may become infected. Between the musculature and the epithelium in the submucous space, are venous plexuses situated one on the left

sites of lesions near the anal orifice are localized as if seen on a clock face with the patient in the lithotomy position. Subsidiary veins occur on either side of the major veins, frequently communicating with a similar plexus under the skin, called the subcutaneous plexus, which drains into the systemic

venous system. Hemorrhoids result from the dilatation of these venous plexuses.

The longitudinal muscle is the division between the *perianal space* and the *ischio-rectal space*. The perianal space lies underneath the skin and contains the subcutaneous sphincter, the external hemorrhoidal plexus, and granular fat. Deep to this is the *ischio-rectal space*, lying between the ischium laterally, and the rectum medially. It contains large globules of fat, loosely packed, and is bounded above by the levator ani muscles. Above the levator ani is the *supra-levator space*, which contains fatty fibrous tissue and the nerves of the pelvic plexus.

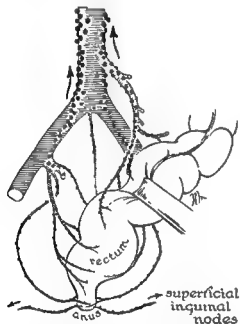


Fig 206—Lymphatic drainage of the rectum

The Lymphatic Drainage of the Anus and Rectum.—The lymph flow from the lower half of the rectal ampulla and the anal canal is to the inguinal lymph nodes. This direction of flow must always be carefully considered in low-lying malignant lesions of the rectum and of course those in the anal canal. The inguinal lymph nodes are also involved in infection of this area.

The flow of lymph from the proximal part of the rectum and the rectosigmoid region

is in a proximal direction, the lymphatics following the course of the blood vessels. Malignant lesions of this part of the rectum, if they spread by lymphatics, nearly always do so in this proximal direction. Retrograde or downward lymphatic spread is likely to occur only if the proximal lymphatic system is blocked by tumor and inflammatory cells. It is therefore possible to anticipate that removal of the entire proximal lymph drainage area in the surgery of a malignant tumor will suffice in nearly every case to remove all lymphatic metastatic areas of spread. It is usually sufficient to remove 2 to 3 cm of rectum distal to the lesion; however, if a low anastomosis is technically difficult, it is safer for the patient, both from the point of view of a leak at the site of anastomosis and from the added insurance of a more complete removal of the tumor, to remove the entire rectum and anus.

Methods of Examination.—Every patient who complains of rectal symptoms, pain, bleeding, constipation, diarrhea, tenesmus, etc., should have a thorough examination. A careful history must be taken. Has there been a change in the bowel habit? Has there been pain at the anal margin? Is there a swelling either externally or internally? Does a mass protrude? Can it be replaced? What is the size of the stool, its color and consistency?

Inspection of the anus with gentle retraction of the buttocks may reveal a sentinel pile, indicating a painful fissure, or a thrombosed external hemorrhoid with its blue, swollen appearance may be seen. In this case further examination must be postponed until after the patient has been anesthetized. Apart from these two painful conditions, digital examination should always be performed. The patient may be postured on the left side, or in the knee-chest position. Gentleness and reassurance are necessary at all times. When the finger has been fully introduced the puborectalis

muscle can be palpated posteriorly and laterally and the tone of the sphincters determined. Even the notch between the superficial and deep external sphincter can usually be felt. Crypts, polyps, and fistulas can be detected. Continuing the rectal examination, the prostate and its consistency is determined in the male, while the pelvic organs are examined in the female. It is convenient to complete bimanual pelvic examination, especially in children and virgins, by this method. Next the finger is swept laterally and posteriorly to examine the coccyx and lower sacrum for mobility, pain or other abnormalities. Internal hemorrhoids cannot be felt. *However, more than half of the carcinomas of the colon and rectum lie within reach of the examining finger.*

Examination should be completed by the use of the anoscope, proctoscope, or sigmoidoscope. Before completing this instrumental examination, swabbing of the mucosa

may be performed for culture of organisms. A biopsy may be taken from an ulcer or growth, or fulguration of a polyp may be carried out through the instrument.

CONGENITAL ABNORMALITIES CONGENITAL STENOSIS

Partial narrowing or stenosis at the junction of the rectum and anal canal may occur as an intermediate stage between normal and imperforate anus

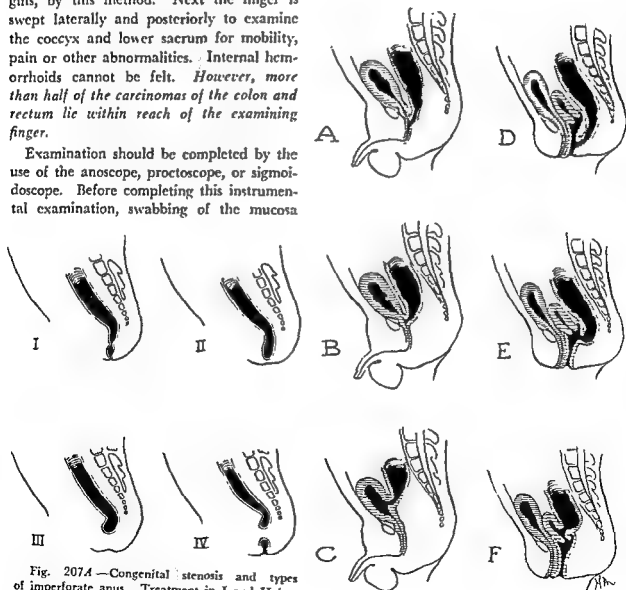


Fig. 207-A—Congenital stenosis and types of imperforate anus. Treatment in I and II is a simple dilatation or a cruciate incision in the anal diaphragm. Treatment in III and IV requires considerable mobilization of the proximal bowel (After Ladd and Gross.)

Fig. 207-B

Types of associated fistula encountered in male patients.

Types of associated fistula encountered in female patients.

IMPERFORATE ANUS

Imperforate anus is a congenital defect of development of the hindgut and proctodeum. The cloaca is divided by the septum transversum which separates the bladder from the hindgut. The hindgut itself becomes canalized and should meet the anal dimple. Failure of this process results in imperforate anus.

There are three main types of imperforate anus.

1. In the first type there is a complete rectum but with a membranous occlusion at the junction with the anal canal.

2. In the second type there is an absence of the anal dimple with the rectum separated by several centimeters from the skin.

3. In the third type there is a patent anal canal with the rectum ending in a blind pouch at some distance from it.

In addition to the atresias, the sexual fistulous types are encountered. In the male, the hindgut may end in the bulb of the urethra, the prostatic urethra, or the bladder. In the female, the rectum may end in communication with the vagina near the orifice or at the vault, or in the uterus itself. The anal dimple may or may not be present in association with these fistulous forms.

Clinical Picture.—The usual clinical picture is one of intestinal obstruction, presenting immediately after birth. Only in the cases with stenosis of the bowel is there lack of this symptom-complex. The imperforate anus should be diagnosed at birth if difficulty is encountered when an attempt is made to insert the rectal thermometer. Further, the absence of meconium will suggest examination of the anal area. At this time, bulging of the thin proctodeal membrane may be noted when the infant cries. The presence of an associated fistula is evidenced by the passage of meconium per urethra or vagina.

A plain x-ray film of the abdomen is an essential diagnostic procedure. It confirms the intestinal obstruction by demonstrating

loops of bowel dilated with air, and it is invaluable in ascertaining the level of the blind end of the rectum. The position of this blind pouch must be known accurately before operative intervention.

Technique of X-ray of Abdomen.—A radiopaque marker is placed over the anal opening or anal dimple and lateral exposures are taken with the child held on his back with the thighs flexed on the abdomen. The lateral exposure is important as it enables a more accurate measurement of the distance from the bowel to the surface marker. This supine posture is preferred to the head down position as the air can fill the distal bowel, yet there is no tendency for the bowel to fall away from the pelvis and so give an exaggerated impression of the distance from rectum to skin.

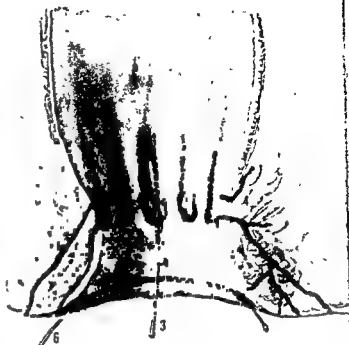
Treatment.—The stenotic cases can be treated by rectal dilatation. The thin membranous diaphragms are easily divided and constitute a simple surgical problem.

If the upper blind pouch is fairly close to the skin edge or anal canal, e.g., 2 cm., it can be approached through a perineal incision and brought down to be sutured to the skin margins of the anal canal.

If the upper blind pouch is high up in the pelvis, a colostomy may be performed, or a combined abdominoperineal approach be made. The sigmoid and descending colon is then freed from its attachment to allow the bowel to be brought down and subsequently stitched to the anal skin margins.

The coexistent vomiting must be prevented by nasogastric suction, the dehydration must be corrected, and blood transfusions are required during the surgical procedure.

The cases with fistula are an urgent problem in the male in order to prevent urinary infection, which may ascend as pyelonephritis. A defunctioning colostomy is imperative. In the female, on the other hand, the fistulous tract serves as a safety vent for the otherwise obstructed bowel. The repair



I.H. Internal Hemorrhoids
 E.H. External Hemorrhoids
 S.T. Skin Tags
 C. Cryptitis
 P. Papillitis

1 and 2. External Fistulae
 3. Complete Internal Fistula
 4. Blind Internal Fistula
 5. Branching Type Fistula
 6. Horseshoe Fistula

F. Netter
 M.D.



RECTAL PROLAPSE



FISSURE-IN-ANO

of the fistula and the adjustment of bowel continuity may be attempted at a slightly older age.

FISSURE-IN-ANO

This painful condition is usually associated with constipation. The hard scybalous mass tears the anal valve usually in the midline posteriorly, i.e., 11 o'clock, down to the anal verge, thus leaving a raw area or fissure with the subcutaneous sphincter exposed. The patient complains of a sharp, stabbing pain on defecation which is caused by spasm of the sphincter. This is often followed by the passage of a few drops of blood. On examination there is an hypertrophied tag of anal tissue (sentinel pile) usually posteriorly, but in about 10% of cases anteriorly, in the base of which may be seen the lower end of the fissure.

The treatment consists in softening the stool by liquid paraffin, and injection of local anesthetic in an oil base which should afford relief of pain for approximately a week. If this fails, then under anesthesia, the sphincter is dilated up to four fingers in size, and the fissure and sentinel pile are excised, usually combined with partial severance of the subcutaneous external sphincter.

HEMORRHOIDS

External hemorrhoids are varicosities of the external hemorrhoidal veins. These form enlargements around the anal margin, and occasionally become thrombosed. The occurrence of this thrombosis is marked by acute pain and the appearance of a firm lump at the anal margin. On examination the overlying skin is bluish in color and there is a firm tender mass, which is nonfluctuant.

Treatment.—The pain and discomfort can be relieved by the moist heat of hot baths or compresses. An incision under local anesthetic will enucleate the dark blue clot and gives immediate and dramatic relief.

Internal hemorrhoids consist of the enlargement of the internal hemorrhoidal ve-

nous plexuses, the main sites being at 3, 7 and 11 o'clock. The hemorrhoidal plexus is one of the main connections between the portal and systemic circulation, the others being the lower end of the esophagus, around the umbilicus, and between the renal and lumbar veins. Patients are predisposed to hemorrhoids by the inheritance of weak walled veins, and also by the absence of valves in the internal hemorrhoidal veins. The predisposition to hemorrhoids is aggravated by an increase in intra-abdominal pressure from straining at stool, constipation, pregnancy and pelvic tumors. Hemorrhoids may also occur in portal hypertension.

Symptoms.—By far the commonest symptom is bleeding, usually at the time of defecation. The patient may also complain of prolapse, that is, the piles protrude and may have to be replaced manually. Discharge and irritation may accompany hemorrhoids, while pain is only associated with the complication of prolapse or thrombosis. They may occur at any time from adolescence onward. On examination the bluish bulging mucosa is readily recognized in the three primary positions, although it may vary in size. The examiner must be careful to distinguish between prolapsing internal hemorrhoids, and prolapse of the rectum. This can be done easily, by asking the patient to strain. If a complete ring of mucosa appears outside the anal margin prolapse of the rectum is obvious.

For convenience in description first degree hemorrhoids are those that bleed but do not prolapse. Second degree cases prolapse and return again with or without bleeding. Third degree piles prolapse and become irreducible.

Complications.—The commonest complication is anemia due to both the amount and duration of bleeding. The second complication is strangulation and thrombosis which occurs only with prolapsing hemorrhoids. Thrombosis may be followed by infection, accompanied by a sharp throbbing pain, or ulceration with a persistent dis-

charge. Fortunately portal pyemia is now practically unknown.

Treatment.—This may be considered from three points of view. In mild cases and young individuals, palliative treatment may be all that is required. This consists of cold compresses combined with an analgesic ointment or suppositories. In those cases in which operative treatment is contraindicated, or in the milder cases, *injection therapy* may be carried out. The actual selection of cases is of fundamental importance in this treatment, the most suitable being first or second degree piles with no fibrosis and no external hemorrhoids. It is an unsuitable method for prolapsed third degree hemorrhoids, for fibrosis of internal piles or for the compound external-internal variety. Injection therapy is carried out with a 5 cc Luer-Lok syringe and a long needle with a shoulder less than one-half inch from the tip. Three milliliters of 5% phenol and 5% menthol in almond oil is injected into the submucosa near the base of the internal hemorrhoid, injecting one at a time at weekly intervals.

Complications of this treatment are remarkably few but abscess formation and sloughing are occasionally encountered. If the injection is placed too low, there will be considerable pain from the phenol solution in contact with the anal sensory nerves.

Operative treatment is indicated in second and third degree hemorrhoids. It is contraindicated in the presence of infection or gangrene and such general conditions as portal hypertension. A sigmoidoscopic examination is performed to exclude any other local organic cause. The hemorrhoids are dissected with a small flap of skin, demonstrating the subcutaneous external sphincter, and leaving a bridge of normal mucosa between the hemorrhoids. The base is ligated by transfixion, the bleeding points are carefully tied, and an absorbable hemostatic agent may be used as a pack. Early ambulation, hot baths, and mineral oil by mouth

help toward a comfortable convalescence. Digital dilatation may be necessary to avoid a stricture. Postoperative pain may be prevented by injecting a local anesthetic in oil into the area at the time of operation.

Postoperative Complications.—Retention of urine is common particularly with the use of spinal anesthesia. Secondary hemorrhage occurs occasionally, but careful technique will minimize this complication. Stricture formation may result if too much tissue is removed at the primary operation. This may often be prevented by repeated dilations in the early postoperative period.

PROLAPSE OF THE RECTUM

Prolapse of the rectum is divided into two types: partial, consisting of the mucous membrane only; and complete, involving the entire thickness of the rectal wall. *Partial prolapse* usually occurs at the extremes of age. In children it is due to a straight sacrum, or to a lack of fat in the ischiorectal fossa. The precipitating causes are usually constipation, straining, or diarrhea. In adults partial prolapse is often associated with hemorrhoids, and always results from straining efforts associated with constipation or enlargement of the prostate. *Complete prolapse* is rare. It is more common in females, occurs in both stout and thin persons, and should be looked upon as a sliding hernia of the pouch of Douglas.

Symptomatology.—There is slight bleeding after straining, and discomfort of a mild type. Complete prolapse may be caused at any time by straining. There is a mucous discharge, incontinence, and regulation of bowel habit is difficult so that the patient usually has to wear some pad or support. On examination, inspection is of the greatest importance. A swab is held over the anus so that there will be no incontinence when the patient is asked to strain. The rectum sometimes protrudes as much as 18 inches. Palpation reveals the loss of sphincter tone. There may be ulceration of the mucosa, and

even strangulation of the protruding mass, leading to irreducibility and gangrene. The diagnosis is easy, but may be missed unless the patient is asked to strain. It is important to see the full extent of the prolapse, to replace the bowel as soon as it comes out, and then to decide on the line of treatment.

Treatment in Children.—This is usually conservative. A regime of bowel training must be instituted, and the child's general health improved. If this fails linear cauterization of the mucosa, or injection of 5% phenol in almond oil, or 95% alcohol into the submucosa is recommended.

Treatment in Adults.—In *partial* prolapse associated with hemorrhoids it is only necessary to do a hemorrhoidectomy. The injection of 5% phenol in almond oil into the submucosa is helpful. In *complete* prolapse of the rectum, operation is advisable. Gabriel advocates the incision of the mucosa around the anal canal with amputation of the prolapsing portion of bowel through a circular incision and subsequent anastomosis. The second method is that of Roscoe Graham. the abdomen is opened, and the floor of the pelvis is reconstructed; the rectum is then sutured to the pelvic floor, thus preventing prolapse from above. Unfortunately both of these methods have a fairly high incidence of recurrence, because the underlying causes are still present.

CRYPTITIS

The small crypts behind the valves of Ball, and between the bases of the columns of Morgagni, frequently collect infected material, and these inflamed pockets are responsible for anal spasm, pruritus, pain on defecation and even frequent bowel movements. The treatment consists of hot sitz baths, and dilatation of the sphincter. The crypts should be exposed by a blunt hook, and the free edge cut to give adequate drainage. The infection not infrequently burrows under the mucosa, to cause a submucosal abscess.

ANORECTAL ABSCESS

Infection may be localized under the mucosa of the rectum, or of the anus, as described in Cryptitis, or the infection may be outside the muscular layer either above or below the levator ani. Cases in which the infection is above are usually due to diverticulitis or ulcerative colitis, while those below originate in the ischio-rectal fossa. These abscesses, and their situation, are extremely important in themselves, but they become more so when considered from the point of view of their complications, of which the main one is fistula-in-ano.

The commonest type of anorectal abscess is the *perianal abscess* which may arise from the crypts, hair follicles, or sweat glands, associated with pruritus or systemic infection. The patient complains of pain on defecation which is persistent and steady, and gradually increases in intensity until he cannot sit, stand, walk, or lie down without pain. The examination is difficult because the sphincter is in spasm and the induration is frequently masked. Examination under anesthetic is necessary to determine the exact site of the abscess, and incision and adequate drainage should be carried out in order to prevent tracking of the abscess and fistula formation.

SUBMUCOUS ABSCESS

The mildest form of anorectal abscess is a submucous collection of pus, usually on the lateral wall of the rectum. It is often caused by abrasions, but may follow thrombosis or injection of hemorrhoids.

Treatment.—These abscesses may rupture by themselves into the rectum or anal canal; otherwise, they should be incised and drained and the edges cut to saucerize the area.

ISCHIORECTAL ABSCESS

The loose cellular tissue of the ischio-rectal fossa may become infected from the rectum or skin, or infection may be brought there by the blood stream. The abscess may communicate with the rectum between the

superficial and deep sphincters. It is very slow in development, with gradually increasing discomfort, deep throbbing pain, malaise, and fever up to 103° F. There are fullness and tenderness on examination both externally and by rectum.

Treatment.—Under anesthesia, a cruciform or T-shaped incision is made, the edges are removed to saucerize the wound, and the cavity is packed. Postoperative sitz baths are instituted twice daily and the wound is allowed to granulate.

PELVIRECTAL ABSCESS

Pelviorectal abscess is the rarest variety and involves the space above the levator ani. Infection usually arises from inside the pelvis. It may rupture into the rectum or through the levator ani. These patients have pelvic pain or discomfort, malaise, fever, and a marked leukocytosis.

Treatment.—The abscess should be drained through the ischiorectal fossa. It should be treated early in order to prevent complications, particularly high fistula-in-ano.

FISTULA-IN-ANO

Fistula-in-ano invariably results from a rectal abscess which burrows its way to the surface, producing a chronic granulating

tract. The track passes between the anal canal and the skin. Fistula-in-ano may be complete or incomplete. The complete variety has a fistulous connection between the skin and rectal lumen: there may be intermittent leakage of pus or feces. The incomplete form discharges either into the lumen of the bowel or through the perianal skin.

Etiology.—It is a disease of adult life, commoner in males. The infection may begin as an abscess in the submucosa and burrow its way to the surface, as an ischiorectal abscess, or more rarely from diverticulitis or ulcerative colitis.

Classification of Fistula-in-Ano.—

1 *Submucosal and Subcutaneous Fistulas.* These occur in about 5% of cases.

2 *Low Anal Fistulas.* Here, the track may be demonstrated by means of a probe passed from the skin to the anal canal, deep to the subcutaneous external sphincter. These make up about 75% of fistulas.

3 *High Anorectal Fistulas.* These usually begin from an ischiorectal abscess which has drained into the rectum as well as externally. They comprise about 10% of cases.

Diagnosis.—

1 *Inspection:* Near the anus there is a small pimple from which pus may be dis-

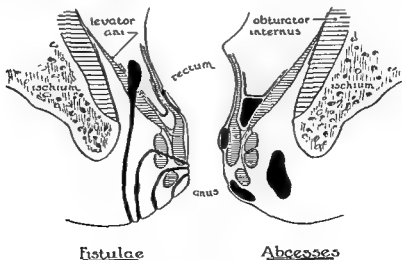


Fig. 208—Fistula-in-ano.

charging. The anterior fistulas are usually single, while those posterior frequently have multiple openings and side tunnels.

2. *Palpation:* On inserting the finger into the rectum and squeezing downward, pus may be extruded, and with the passage of a probe, the tract may be outlined. Should one suspect a complicated fistula, the injection of radiopaque Lipiodol is of assistance in estimating the complexity in a given case.

The diagnosis of fistula-in-ano presents little difficulty. The important point is to establish the bacteriological cause, whether the lesion is due to pyogenic organisms or due to the tubercle bacillus or actinomycetes. In tuberculosis there is usually a history of pulmonary infection, and if this is suspected, the specimen must be examined bacteriologically and pathologically. Actinomycosis is rare in this region and is diagnosed in a similar manner.

The prognosis depends on the level of the fistulous tract and the etiological agent.

Treatment.—

1. *Submucosal and Subcutaneous Fistulas.* These should be completely excised through their whole length and allowed to heal by granulation.

2. *Low Anal Fistulas.* Treatment consists in careful exploration with the probe and complete excision of the tract, cutting the subcutaneous sphincter in one place only.

3. *High Anorectal Fistulas.* The important point here is the level of the communication into the anal canal or rectum. It may be

(i) Between the superficial and deep external sphincter.

(ii) Between the deep external sphincter and the levator ani.

(iii) Above the levator ani

Treatment of the high level anorectal fistulas constitutes an extremely difficult surgical problem. When the fistula extends between the superficial and deep external sphincter, it is possible to excise the tract and preserve the deep portion of the exter-

nal sphincter, thus maintaining adequate continence. When, however, the fistula is above the external sphincter, it is necessary to resort to staged surgery. The first stage consists of wide excision of the tract with division of the superficial external sphincter, and the preservation of the deep portion. A heavy silk suture is tied around the remaining internal sphincter. The second stage is then carried out two or three weeks later. The suture prevents retraction of the sphincter because of the resultant surrounding fibrosis. The remainder of the tract is then excised.

PRURITUS ANI OR PERIANAL DERMATITIS

Local irritation of the skin around the anus may come on at any age. There are many causes: in children it is usually due to pinworms and general lack of cleanliness. In men it is frequently associated with excessive sweating. Some patients may have a relaxed sphincter with a continual leak of mucus. Others have excoriation of the skin which is heaped up in folds, with small fissures, or ulcers between the folds. This is a warm, moist area suitable for the growth of parasites, and mycotic infection. Lastly, there are those of obscure origin due to allergy or even psychoneurosis.

The investigation of a case in which the patient complains of itchiness in the anal region demands a thorough history and general examination, as well as a careful local examination. It is advisable to look especially for crypts, excoriation of the skin, and any other local cause, as well as careful examination for fungi, etc. The treatment is directed toward:

1. Hygienic measures. Sitz-baths twice daily, a dry dusting powder, clean, well-fitting underclothes and clean bed linen.

2. Dietetic measures. If diet is a suspected etiological agent, various elimination procedures may be tried.

3. Medical therapeutics. Treatment of constipation by mild laxatives such as liquid

paraffin, and the attendance to nutritional regimes

4 Surgical treatment is reserved for intractable cases, and consists in careful preparation of the patient for 2 or 3 weeks before admission with local antiseptics such as methylene blue or potassium permanganate. The definitive operative therapy provides wide excision of the skin and all the redundant folds in either a butterfly shape or sunflower pattern. This cuts the cutaneous nerves, and enables the denuded areas to heal by granulation tissue and become smoothly epithelized

PROCTITIS

Proctitis is a comprehensive term, including all the types of inflammation of the mucosa of the rectum. Simple, catarrhal, non-specific proctitis is a localized form of colitis. It may be due to vitamin deficiencies, endocrine dysfunction, allergy, vasomotor disturbances, or even psychological causes.

Nonspecific

Acute proctitis is an acute hyperemia of the rectal mucosa with edema, vasodilatation, and even hemorrhage. There is a granular appearance of the mucosa, with small, pinpoint ulcers. Digital examination of the rectum reveals that the wall is thickened and the lumen narrowed. The proctoscope shows a red, granular mucosa, which bleeds easily. Bacteriological culture may reveal streptococci, staphylococci, and hemolytic streptococci as well as *B. coli*

Chronic proctitis is the more chronic form of the disease, and may be either hypertrophic or atrophic.

Irradiation proctitis occurs after deep x-ray therapy to the pelvis, or radium treatment for carcinoma of the cervix. It consists of a hyperemic, swollen mucous membrane with excess of mucus, and it is usually accompanied by rather severe steady pain. General treatment consists of liquid paraffin for soft stool formation, and cleansing

enemas. In more severe cases, a colostomy may be necessary for the relief of the recurring hemorrhage or stenosis.

Specific

Bacillary or Amebic Dysentery must be confirmed by the growth and recognition of the causative organism.

Tuberculous proctitis is usually associated with an active pulmonary lesion, and may be complicated by tuberculous anal fistulas.

Gonococcal proctitis is an acute proctitis with hyperemia of the mucosa and thick creamy pus. It must be bacteriologically diagnosed.

Syphilitic proctitis. A primary chancre in the rectum may occur and usually presents as a small ulcer.

Venereal proctitis is a lymphogranuloma venereum which may appear as an abscess, a stricture, or a small ulcer.

Treatment.—The treatment has been greatly simplified since the introduction of antibiotics. General medical measures may be used, such as sitz-baths, oil enemas and soothing ointments. The nonabsorbable sulfonamides will usually control acute non-specific proctitis, while penicillin is specific for gonorrheal and syphilitic proctitis. Tuberculous proctitis is much more difficult to treat, demanding both general and local care. Streptomycin or its kindred antibiotics are efficacious for lymphogranuloma venereum.

INJURIES OF THE RECTUM

Injuries of the rectum are not very common in civilian practice, but increase during wartime. They may be divided into two classes—operative and traumatic. Accidents to the rectum during operative procedures occur during operations on the urethra, prostate, vagina, or uterus. Perforations are also caused by the sigmoidoscope or enema nozzles, which may perforate the rectum. A surrounding cellulitis or peritonitis may result. Recognition of such injuries usually is made at the time of the accident, and the

opening into the rectum can then be repaired immediately.

War Injuries.—Gunshot wounds of the pelvis may affect the intra- or extraperitoneal portion of the rectum.

In crush injuries of the pelvis, a spicule of bone may perforate the rectum.

Rectal perforation also occurs from swallowed foreign bodies or from those introduced from below. These range all the way from a swallowed bone which has lodged transversely in the anal canal, to the infinite variety of objects inserted into the rectum by children and lunatics. After their removal the rectum should be examined to make certain that there has been no damage done to the wall. An anesthetic is frequently necessary or otherwise the procedure is extremely painful.

STRICTURE OF THE RECTUM

Congenital stricture has been mentioned in the section on imperforate anus. Post-traumatic stricture may follow perforating wounds of the rectum, and postoperative stricture may occur after hemorrhoidectomy or low intestinal anastomosis. Irradiation proctitis may result in a stricture formation. Lymphogranuloma inguinale is a common cause of stricture formation. This is a venereal infection caused by a filterable virus, and can be diagnosed by the Frei antigen test. Local examination of the rectum reveals a definite fibrous stricture which is usually long and tubular associated with perirectal inflammation. In all cases of stricture the patient complains of progressive difficulty of defecation, straining at stool, and ribbon-like stools.

Treatment.—

1 *Dilatation* is done repeatedly using rectal bougies of graduated sizes.

2. *Operation.* Internal proctotomy consists in cutting the fibrous stricture in 5 or 6 places.

3 The patient may require a *colostomy*, usually on the left side. Many cases of

benign stricture of the rectum may be prevented by the early use of the antibiotics in the treatment of the specific cause.

4. The extensive stricture of lymphogranuloma inguinale may require an abdominoperineal resection.

BENIGN TUMORS OF THE RECTUM

The commonest tumor of the rectum is a benign adenoma. The connective tissue tumors such as lipoma, fibroma, myoma, lymphoma, and angioma are rare. An adenoma is a small swelling, usually about the size of a pea which later enlarges up to about three centimeters in diameter. It may be sessile or pedunculated and may be the site of malignant changes. The usual symptomatology is bleeding, discharge, occasionally constipation, or diarrhea. In children the adenomas which usually cause bleeding, are single, and after removal cause no further trouble. In adults, the adenomas vary in size, number, and situation, and in symptomatology. Diagnosis is confirmed by sigmoidoscopic examination and visualization of the polyp. It can then be grasped by forceps and removed by either a snare or cautery. The only point that should be stressed is that if there is an hereditary influence, or when the papillomas are multiple, then it is wise to have a radiographic examination in order to be sure that they do not extend along the bowel. Multiple polyposis of the colon, no matter what the age, is definitely a premalignant condition, and treatment is total colectomy. (See Familial polyposis.) Anal warts or multiple papillomas of the skin around the anus occur. Treatment consists in removal by scissors under local anesthesia. The radiating wounds are then packed with petrolatum gauze.

CARCINOMA OF THE RECTUM

Incidence.—Cancer of the rectum and lower sigmoid comprises approximately 7%

of all malignant tumors of the body. Men are affected more commonly than women in the ratio of 3 to 2. While no age is exempt, the most common decade for the occurrence of this form of cancer is 50 to 60, although many cases have been reported under the age of thirty.

Etiology.—Though the true etiology of cancer is as yet unknown, certain lesions may be considered premalignant in the rectum. This is particularly true with multiple or single polyps.

Pathology.—A carcinoma of the rectum, excluding that of the anal canal, is an adenocarcinoma. It is in its early stages a local disease which begins as a local change in the epithelium or as a malignant change in a preexisting adenoma. Varieties of adenocarcinoma which have been described include: medullary, scirrhous, mucoid, and papillomatous.

Of more importance is the microscopic grading of the neoplasms, as has been described by Broder, who has divided them into 4 groups according to the degree of differentiation of the cells and the number of mitoses. The more undifferentiated the cells, the more malignant the tumor.

Malignant lesions of the rectum spread by direct extension, venous and lymphatic channels. It has been pointed out by Dukes that carcinoma of the rectum extends by direct spread through the rectal wall, and that lymphatic spread does not take place until the growth has reached the extrarectal tissues. The intramural lymphatics cause a spread within the wall of the bowel but for only a short distance above or below the lesion.

Of much more importance are the extramural lymphatic channels. Three methods of extension occur: (1) downward, (2) laterally along the levator ani muscles, (3) upward to the retrorectal glands and then along the superior hemorrhoidal and inferior mesenteric vessels toward the periaortic glands.

Venous spread, while much more serious than lymphatic involvement, occurs in 20% of cases.

Symptoms.—Carcinoma of the rectum in its early stages gives symptoms which may be slight or even absent, and the lesion may occasionally be discovered on routine physical examination. However, the most frequently encountered symptoms are bleeding, alteration in bowel habits and pain.

Blood in small amounts accompanied as a rule by the passage of excessive mucus in the stool is the most common complaint. The blood is bright red and may either be mixed with the stool or may be streaked on the surface. The actual total blood loss is small, and anemia is not as marked as with carcinoma of other parts of the colon.

Alteration in bowel habit may be the earliest symptom. This may take the form of constipation or diarrhea. Tenesmus may be a prominent feature.

Pain is a late symptom and denotes a far advanced malignancy.

It is obvious that none of these symptoms are diagnostic of rectal cancer, but their presence should direct one's attention to a thorough examination of the rectum and colon.

Treatment.—The diagnosis having been established by biopsy, choice of treatment will depend on the site and size of the tumor. The results of radiation therapy have been disappointing, so that the only treatment is surgical removal.

For the noninvasive carcinoma confined to the mucosa and for carcinoma-in-situ, particularly in a polyp in the rectum which is within reach of the proctoscope, local excision with regular follow-up examinations afterward is a safe treatment. Invasive carcinoma, no matter how small, must be treated by radical excision. Carcinomas situated above the peritoneal reflection are usually classified clinically as situated in the rectosigmoid. In these cases, if the patient is not too fat, or the pelvis too small, it is

possible to do a resection with end-to-end anastomosis. When the growth is at the level of the peritoneal reflection and below, then it is wiser to perform the abdominoperineal resection of Miles with terminal colostomy. This operation has recently been modified by the use of the two team synchronized combined abdominoperineal operation. The advantage of this method is wider excision; and more extensive growths can be removed because direct vision from above and below at the same time is possible. Some authors advocate a proctosigmoidectomy with preservation of the anal sphincter. This method is suitable for selected cases, Stages I and II only, with the growth at least three inches above the anal margin, and where the architecture of the sigmoidal arteries permits adequate mobilization of the sigmoid down to the anus. For advanced cases there is considerable debate whether abdominoperineal operation should be performed when metastases are present in the liver. However, if the local growth can be removed, the last days of the patient will be more comfortable. In obstructed cases the obstruction must be relieved by means of a proximal colostomy, followed at a later date by abdominoperineal resection

becomes blocked, it may go on to cyst formation. It may contain hair and retained secretions, and inflammatory changes frequently occur. The sinus opening may be single or multiple.

It is thought to arise from faulty development in the medullary canal or failure of union of the median raphe in this region

Clinical Features.—In childhood this condition is asymptomatic and consists of one or more small dimples in the midline. Symptoms do not as a rule develop until young adult life and are due to infection in the sinus which intermittently closes, fills up and discharges again. Occasionally infection may spread and break out causing new sinus openings. This may be precipitated by trauma such as falls on the buttocks. It is more common in males.

Treatment.—In the acute stage abscesses should be drained. During the quiescent period all diseased tissue should be excised. If the resected area is not too large, the skin edges should be approximated, care being taken to obliterate all the dead space.

So-called recurrences are due to inadequate primary removal of the sinus tract.

CARCINOMA OF THE ANUS

Carcinoma of the anus is a rarer disease than carcinoma of the rectum. It is of squamous-celled type, and produces early stenosis of the canal. These behave like cutaneous growths with secondaries in the inguinal glands and may be treated by both surgery and radiation therapy. Malignant melanoma also occurs, and responds only to radical surgery.

PILONIDAL SINUS (SACRO-COCCYGEAL SINUS)

Pilonidal sinus is a sinus lined with stratified squamous epithelium which is situated in the midline in the region of the sacro-coccygeal junction. When the sinus tract

REFERENCES

- Allen, A. W., Welch, C. E., Donaldson, G. A.: Carcinoma of the Colon, *Ann. Surg.* 126: 19-30, July, 1947.
- Babcock, W. Wayne: Radical Single Stage Extirpation for Cancer of the Large Bowel, With Retained Functional Anus, *Surg., Gynec. & Obst.* 85: 1-7, July, 1947.
- Colcock, Bentley, P.: Prognosis in Carcinoma of the Colon and Rectum, a 10 Year Follow-Up of 337 Patients, *Surg., Gynec. & Obst.* 85: 8-13, July, 1947.
- Coller, F. A., and Ransom, H. K.: Carcinoma of the Rectum. Conclusions Based on 12 Years' Experience With Combined Abdominoperineal Resection, *Surg., Gynec. & Obst.* 78: 304-315, March, 1944.
- Dukes, C. E.: Cancer of the Rectum: Analysis of 1,000 Cases, *J. Path. & Bact.* 50: 527-539, May, 1940.
- Dukes, C. E.: The Classification of Cancer of the Rectum, *J. Path. & Bact.* 35: 323-332, May, 1932.
- Gabriel, W. B., Dukes, C. E., and Bussey, H. J. R.: Lymphatic Spread in Cancer of the Rectum, *Brit. J. Surg.* 23: 395-413, Feb., 1935.

- Gabriel, William B.: *The Principles and Practice of Rectal Surgery*, ed 3, London, 1945, H K Lewis & Co, Ltd
- Gilchrist, R. K., and David, V. C.: Lymphatic Spread of Carcinoma of the Rectum, *Ann. Surg.* 108: 621-642, Oct., 1938
- Gilchrist, R. K., and David, V. C.: A Consideration of Pathologic Factors Influencing Five-Year Survival in Radical Resection of the Large Bowel and Rectum for Carcinoma, *Ann. Surg.* 126: 421-438, Oct., 1947
- Glover, R. P., and Waugh, John M.: The Retrograde Lymphatic Spread of Carcinoma of the "Rectosigmoid Region," *Surg., Gynec. & Obst.* 80: 434-448, April, 1945.
- Grinnell, R. S.: The Lymphatic and Venous Spread of Carcinoma of the Rectum, *Ann. Surg.* 116: 200-215, Aug., 1912
- Ladd, William, E., and Gross, Robert, E.: *Abdominal Surgery of Infancy and Childhood*, Philadelphia, 1941, W B Saunders Company
- McKittrick, L. S.: Principles Old and New of Resection of the Colon for Carcinoma, *Surg., Gynec. & Obst.* 87: 15-25, 1948.
- Maignot, Rodney: *Abdominal Operations*, ed 2, New York, 1948, Appleton-Century-Crofts Company, Inc
- Miles, W. E.: *Cancer of the Rectum*, London, 1926, Harrison & Sons
- Rankin, Fred W., and Graham, A. Stephens: *Cancer of the Colon and Rectum*, ed 2, Springfield, Ill., 1950, Charles C Thomas
- Wangensteen, O. H.: Primary Resection (Closed Anastomosis) of the Rectal Ampulla for Malignancy With Preservation of Sphincteric Function, *Surg., Gynec. & Obst.* 81: 1-24, July, 1945
- Wangensteen, O. H., and Toon, R. W.: Primary Resection of the Colon and Rectum With Particular Reference to Cancer and Ulcerative Colitis, *Am. J. Surg.* 75: 384-404, Feb., 1948

CHAPTER XXIX

ABDOMINAL ASPECTS OF GYNECOLOGY

G. A. SIMPSON, M.D.

When the patient seeks advice for lower abdominal pain, vaginal bleeding, or disturbances of micturition, the physician's attention is directed to the pelvic organs. A careful history and a bimanual pelvic examination enable him to make a tentative diagnosis and to initiate the proper investigation. Once the true diagnosis is apparent treatment may be instituted. Other diseases of the female pelvic organs create clinical syndromes which simulate those of acute and chronic surgical conditions. To aid the general surgeon, the clinical significance of the symptoms and signs of these gynecological disorders will be stressed, especially with reference to the differential diagnosis of lower abdominal pain or masses

THE CLINICAL EXAMINATION

Correlation of the patient's history with the abdominal and pelvic examination is a matter of experience. If pelvic examination be made a routine procedure, one soon appreciates the presence of abnormal tenderness or masses.

The bimanual examination is done with method. The patient should be in the lithotomy position on a table. It is imperative that the bladder and lower bowel be empty. Speculum examination of the cervix and vaginal walls should be routine. If cytology studies are to be done the smears should be taken before the parts are covered with lubricant. A rectal and rectovaginal examination should be made if the vaginal findings are obscure.

Again one should make a practice of always performing a bimanual examination on the anesthetized patient about to have a gynecological operation. If the abdomen be

then opened, one has an immediate visual check on the pelvic organs.

If the patient must be examined in bed, it is more convenient to examine the cervix and vagina with a Sims' speculum with the patient in the left lateral position with the hips well to the edge of the bed. When the patient has been confined to bed for any period there will always be an increased amount of retained secretion so that a gentle vaginal toilet is necessary.

PAIN DUE TO PELVIC DISEASE

Pain of pelvic origin may be diffuse over the whole lower abdomen; more commonly, it is described as being worse in one or other lower quadrants, usually just above Poupart's ligament. However, the patient is often very indefinite about its severity and location and she is disturbed mostly by the chronicity of the discomfort.

The pain tolerance of the individual is sometimes difficult to assess, and this difficulty is increased because pelvic pain may be present to a disabling degree with very little evidence of pelvic disease. Conversely

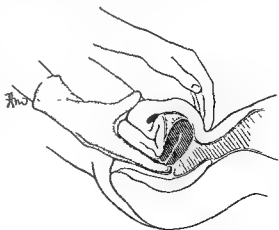


Fig 209.—Bimanual pelvic examination

a rather large pelvic mass may be present without causing symptoms. Interest has recently been revived in the clinical syndrome caused by chronic pelvic congestion. In this, pain is one of the most characteristic symptoms, and the degree of tenderness is quite out of keeping with the pelvic findings.

The relation of the pain to the menses is important. Chronic pelvic inflammatory disease usually flares up in the postmenstrual phase; midcycle discomfort may be due to ovulation; and in ectopic pregnancy there is the history of a delayed, abnormal or missed period.

Intense, acute, lower abdominal pain is associated with acute salpingo-oophoritis, ectopic pregnancy with rupture, torsion of the pedicle of an ovarian cyst, or degeneration of a fibroma. Except in the presence of continued internal hemorrhage, there is no marked deterioration in the patient's condition and it may, in fact, gradually improve. This is unlike the course of an acute appendicitis, a ruptured viscus or a spreading peritonitis.

Colicky pain suggests a threatened or inevitable abortion, tubal abortion, or the presence of a large uterine polyp. Vaginal bleeding is almost always present with any of these conditions, and serves to exclude lesions of the gastrointestinal or genitourinary tract such as constipation, intestinal obstruction or ureteric calculi.

Dull lower abdominal pain may be caused by the pressure of pelvic tumors and in this instance the discomfort is eased by change in posture. If the tumor is malignant, metastases and local infiltration will cause persistent pain.

Pain in the back in women is commonly caused by conditions other than pelvic disease. Backache, if due to gynecological conditions, is usually in the lumbar or sacral areas. Sacropubic hernia or prolapse of the uterus causes this type of backache with an associated vague discomfort in both groins. With other pelvic disease backache is a late

symptom and the condition is obvious on bimanual examination. Chronic cervicitis with a minimal parametritis may cause this persistent low back pain. Uncomplicated retroversion of a normal, mobile uterus seldom produces symptoms.

ACUTE DISEASE OF THE FEMALE PELVIC ORGANS

An acute abdominal condition in a female may be due to any of the causes common to both male and female which, more properly, are straightforward surgical conditions.

Those common only to the female may usually be differentiated by correlating careful menstrual, obstetrical and gynecological histories with the information obtained by bimanual examination.

Acute salpingitis with pelvic peritonitis, pelvic cellulitis, an ectopic pregnancy with intraperitoneal hemorrhage, complications arising from an ovarian cyst or a fibroma, must be differentiated from acute appendicitis, acute pyelitis, or renal calculi, and occasionally diverticulitis with rupture. A patient with a perforated peptic ulcer is rarely admitted to a gynecology ward, but cases of pelvic inflammatory disease or of ectopic pregnancy may be found on the general surgical service.

Acute Salpingo-oophoritis With Pelvic Peritonitis. Pelvic inflammatory disease is predominantly due to an ascending infection and therefore there is a history of a recent lower tract gonorrheal infection, an abortion, a curettage or a delivery at or near term. The exception is pelvic tuberculosis, but in this condition there is frequently a history of a known primary focus and the symptoms are vague and of long standing. Pneumococcal infections are rare.

It is most important to exclude an acute appendicitis. With pelvic inflammatory disease, the differential points are: the history, the lack of gastrointestinal symptoms, the course of the attack with bilateral pain, the absence of toxemia despite the high

temperature and rapid pulse, and the bilateral lower abdominal tenderness. On pelvic examination, there may be evidence of a lower tract infection, an abortion or a recent delivery, and there is marked tenderness with diffuse bilateral masses present. A rectovaginal examination is valuable when splinting of the abdominal wall precludes a proper bimanual examination.

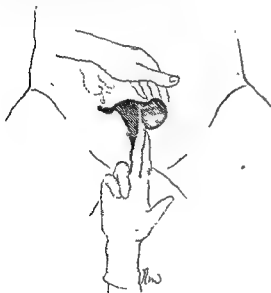


Fig. 210.—Bimanual pelvic examination for pyosalpinx

Pelvic Cellulitis and Pelvic Peritonitis can scarcely be differentiated since both are frequently present at the same time. The inflammatory reaction is most evident in the broad ligaments and cellular tissues. On examination the pelvic organs can scarcely be identified because of the boardlike induration which appears to fill the pelvic cavity. This is the "Plaster of Paris" pelvis of older writers. A lower abdominal mass is formed by inflammatory adhesion of the uterus, bowel, and omentum.

Pelvic Abscess is usually a late (and uncommon) complication of pelvic inflammatory disease. An abscess may form in the midst of the pelvic mass, and this is suspected from the condition of the patient. She remains very ill, with a septic fever and a rapid pulse. There is persistence or re-

crudescence of pelvic pain despite continued bed rest and therapy. Eventually the abscess may point into the pouch of Douglas, the rectum or bladder, or, less commonly, upward toward the peritoneal cavity. If the latter is suspected, rupture of the abscess into the peritoneal cavity must be avoided, and drainage of the abscess through an abdominal incision is imperative. Otherwise the treatment is conservative as long as there is evidence that the abscess is localizing.

Diverticulitis with rupture simulates pelvic inflammatory disease very closely. The diagnosis may not be possible until investigations are completed after the subsidence of the acute symptoms.

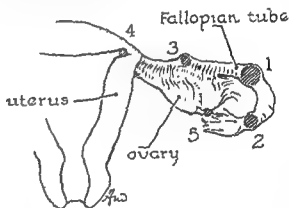


Fig. 211.—Sites and relative frequency of ectopic pregnancy.

Ectopic Pregnancy with rupture and diffuse intraperitoneal bleeding creates the classical syndrome of: amenorrhea of several weeks' duration; sudden, severe, stable lower abdominal pain; and collapse, with all the signs of internal hemorrhage. On examination the patient's pallor is marked and she is in a state of collapse. The abdomen is tender, usually soft, perhaps distended, on pelvic examination there is almost always slight vaginal bleeding and exquisite pain on manipulation of the vault of the vagina or cervix. The cervix is soft and the uterus, if palpable, is enlarged.

With tubal abortion and slower hemorrhage, the clinical picture is less spectacular. The diagnosis is not commonly made on the

first examination. There is an indefinite history of a missed period, then intermittent lower abdominal pain develops, and abdominal tenderness is present. Vaginal bleeding occurs and often persists, though the symptoms and signs become less marked. Often the patient is treated for pelvic inflammatory disease or an incomplete abortion until, later, further internal hemorrhage occurs despite bed rest. Then there is a recurrence of the colicky pain, an increasing anemia develops, and on pelvic examination an indefinite tender mass, a pelvic hematocele, is felt in the pouch of Douglas or in relation to the appendages. Laparotomy discloses the true diagnosis.

Aids to the differential diagnosis of ectopic pregnancy are a pregnancy test, an examination under an anesthetic, curettage, and posterior colpotomy.

Acute appendicitis is excluded by the sudden onset, the presence of uterine bleeding, and the evidence of internal hemorrhage with the associated vaginal findings.

An incomplete abortion creates more profuse bleeding and less pain. On examination there is more marked cervical dilatation. The products of conception, sooner or later, are expelled through the cervix.

An Ovarian Cyst With Torsion of the Pedicle causes severe abdominal pain and peritonismus which may simulate an acute appendicitis or, rarely, a ruptured peptic ulcer. Ordinarily the cyst is of moderate size but enlarges rapidly after torsion and intracystic hemorrhage. The patient may be in a state of collapse with a subnormal temperature. If the tenderness of the abdomen excludes proper abdominal palpation, it is sometimes possible to feel the rounded, tender mass in the pelvis. Examination under anesthesia will verify the diagnosis. Rupture or infection of an ovarian cyst is less common and produces peritonitis. Diagnosis may be difficult. The treatment is laparotomy and removal of the cyst.

Rupture of a Graafian Follicle at Ovulation Time may be painful if minor hemor-

rhage occurs; the bleeding is usually minimal, the acute episode passes, and while regional tenderness may persist, the progressive signs which suggest an ectopic pregnancy with tubal rupture, or acute appendicitis, do not develop.

CHRONIC DISEASE OF THE FEMALE PELVIC ORGANS

Chronic Pelvic Inflammatory Disease. When pelvic inflammatory disease becomes chronic, there is always tuboovarian involvement with adhesion formation which may involve the omentum, the large bowel, and perhaps the small bowel. Chronic pelvic pain is the most common symptom and there may be associated menstrual irregularities. On bimanual examination, the uterus is displaced and the masses may be ill-defined, and often fixed. It is amazing how extensive the adhesions may be when the examination apparently indicates only some bilateral thickening with perhaps impairment of uterine mobility. It is unusual for the disease to be unilateral, though one or the other side may be involved more extensively. Rectovaginal examination is important in this type of pelvic condition since one or other of the diseased appendages is often bound down in the pouch of Douglas.

Pelvic endometriosis, an organized pelvic hematocele, or a diverticulitis of the bowel will simulate chronic pelvic inflammatory disease. Fibromyoma of the uterus may co-exist and cause difficulty in diagnosis.

If there is an acute exacerbation of the chronic condition, the pelvis may seem to be almost completely filled by the inflammatory exudate, the adhesions with omental and bowel involvement are marked. The upper limit of the mass thus formed may extend well above the pelvic brim without creating downward bulging of the pouch of Douglas. It is in this type of lesion that the rectal lumen is constricted and suggests a possible rectal condition.

Pelvic Endometriosis.—Endometriosis implies the presence of endometrium in tissues to which it is foreign. The aberrant endometrium undergoes cyclic menstrual hemorrhage with a resultant chemical peritonitis and this explains the exacerbation of symptoms which arises at the time of the menses. Eventually dense adhesions and fibrosis are produced with encysted collections of degen-

crease in severity as the invasion spreads. There is present the symptom-complex of increasing menstrual pain, dyspareunia, and discomfort due to pressure. Menstrual irregularities may be present and a suggestive feature is a prolonged period of infertility.

In the early stage, pelvic examination discloses nothing palpably abnormal, though thickening of the uterosacral ligaments may

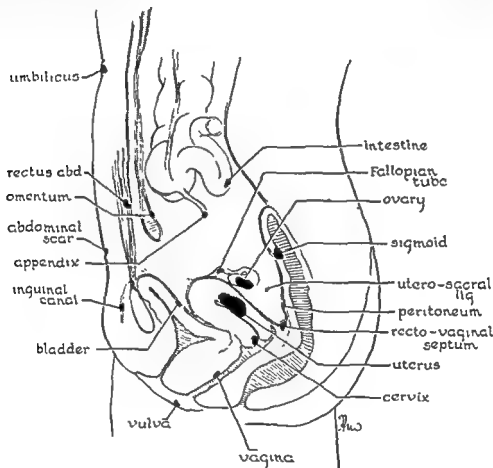


Fig. 212.—Possible locations of endometriosis.

erated blood—*chocolate cysts*. Endometriosis of the pelvic organs is the most common form; however, the large bowel and the bladder may be involved, usually secondarily to the pelvic condition. More rarely extra-pelvic tissues are involved.

Pelvic endometriosis may occur at any time during menstrual life. Chronic symptoms develop which are aggravated with the onset of each menstruation and these in-

be noted. Later, the findings are almost indistinguishable from those of chronic pelvic inflammatory disease. There is, however, no history of fever or previous infection. As the disease extends, fixation of the uterus and bilateral pelvic masses are evident on bimanual examination. At operation minute purplish spots on the pelvic peritoneum or ovaries are apparent, and the mass or masses are found to be chocolate cysts.

The wall of the large bowel may be invaded by endometrial implants which cause fibrosis, and later stricture of the bowel lumen. Abdominal pain and constipation recur with the menses, and eventually partial obstruction may result. Neoplasm of the colon may be suspected, but the rectal mucosa is usually intact, and this serves as a diagnostic aid in excluding carcinoma of the bowel. The bladder wall is not often sufficiently involved to cause characteristic cyclic symptoms and signs.

While removal of the ovaries causes regression, it is preferable in the woman below forty to be conservative. Pelvic tissues which are not grossly involved are not removed and as much ovarian tissue as possible is preserved.

PELVIC TUMORS

Excluding the pregnant uterus at or near term, neglected fibromyomas, very large ovarian cysts, tuberculosis of the peritoneum, or malignant ovarian cysts with metastases and ascites, pelvic tumors do not cause generalized enlargement of the abdomen. Obesity, ascites, and phantom tumors should also be remembered. A full bladder or a loaded bowel is easily excluded by catheterization and emptying the lower bowel with an enema.

At puberty, a lower abdominal tumor may be formed if a persistent vaginal septum—an imperforate hymen—be present and the menstrual fluid accumulates in the vagina (a hematocolpos) and the uterus (a hematometra). The history is that of cyclic, monthly attacks of pain in an adolescent girl who has not menstruated; examination reveals the bulging vaginal septum.

Abdominal distention of a greater or lesser degree is a common complaint at the menopause. The distention comes and goes, and its very indefiniteness ordinarily indicates its minor importance.

A tumor localized to the lower abdomen may be uterine or ovarian in origin. Preg-

nancy, fibromyoma of the uterus, or ovarian cysts are the most common conditions which produce an abdominopelvic mass. It must be remembered that pregnancy and fibromyoma or a pregnancy and ovarian cyst may coexist.



Fig. 213—Positions of uterine myomas

The pregnant uterus is the most common pelvic tumor. The history of amenorrhea, associated with breast changes and the other symptoms and signs of pregnancy should prevent errors of diagnosis. The pregnant uterus is ordinarily uniformly enlarged, and is soft on bimanual palpation. A pregnancy test may be necessary. Otherwise one can postpone the diagnosis until a further month has elapsed, then the rapid, symmetrical enlargement due to the pregnancy will be apparent. Later, of course, fetal movements, fetal heart sounds, or x-ray demonstration of fetal bones give the diagnosis absolutely.

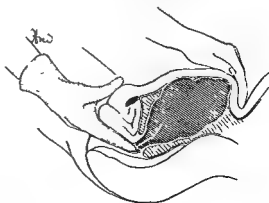


Fig. 214—Bimanual examination for fibroid uterus.

The physician must remember the value of auscultation of the tumor, an x-ray of the abdomen, and a pregnancy test.

Fibromyomas cause enlargement of the uterus, and profuse or prolonged periods are often the chief complaint. Pressure symptoms and pelvic discomfort may be present.

On bimanual examination, the mass is continuous with the cervix, and the uterus is enlarged, usually in an asymmetrical or irregular fashion. Mobility is not impaired unless the fibromyoma is impacted in the pelvis, extends into the broad ligament, or is in association with pelvic inflammatory disease or pelvic endometriosis. No fetal parts or movements are apparent. The mass is dull to percussion and on auscultation a souffle may be present. The pregnancy test is negative and the fibroids grow more slowly than a pregnant uterus.

In certain cases the use of Lipiodol to visualize the interior of the uterus may be an aid in diagnosis. The cavity is usually asymmetrical if fibromyomas are present.

Carcinoma of the fundus ordinarily causes postmenopausal bleeding and, even if there is also enlargement of the uterus, diagnosis cannot be made without seeing the endometrium. For this reason, all uteri removed must be opened at once by an assistant and carefully examined.

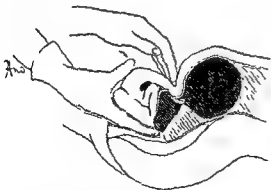


Fig 215—Bimanual examination for ovarian cyst.

Ovarian tumors may be cystic or solid, unilateral or bilateral, on a pedicle or fixed in the broad ligament. Menstrual irregu-

larities may be present, but usually the patient seeks advice because of abdominal enlargement and pressure symptoms. Acute pain occurs only if there has been torsion of the pedicle or intracystic hemorrhage. On examination the breast changes of pregnancy are absent. If the mobile mass is above the pelvic brim on a pedicle, it becomes a midline tumor which commonly feels cystic; the lower end may not be differentiated. The mass is dull to percussion and quite silent on auscultation; i.e., no souffle or fetal heart sounds are present. On bimanual examination, the cervix and uterus are felt distinct from the swelling and pushed out of their proper alignment. The ovarian mass or masses are separate and to one side or in the pouch of Douglas. The pregnancy test is negative. Radiological examination may show the tumor outline.

Too often malignant tumors are not apparent until the abdomen has been opened. The history of rapid growth, the debility of the patient, the pain, the presence of massive ascites, fixation of the irregular hard mass, with deposits in the pouch of Douglas, are suggestive points in the diagnosis.

It should be noted that the ovary may be involved by metastases from carcinoma of other organs, e.g., carcinoma of stomach, colon, or breast. In such cases both ovaries are more commonly involved.

ABDOMINAL PAIN IN PREGNANCY

Abdominal discomfort or pain is a very common complaint during pregnancy. In most instances this is fleeting in character and not of serious consequence. There are other pains, however, which cause more distress. While most of these pains are peculiar

we emphasized that a pregnant woman may develop any acute surgical condition. Furthermore, because of the stretching of the abdominal wall and displacement of abdominal organs due to the enlarged uterus,

the classical symptoms and signs may be obscured and the true diagnosis delayed.

An important point has been stressed about pyrexia when one is dealing with acute abdominal pain in pregnancy. It has been observed that the onset of abdominal pain associated with pyrexia is due to some conditions outside the pregnant uterus; but the absence of early pyrexia does not, of course, exclude extrauterine lesions either obstetrical or surgical. An infected abortion and a degenerating fibroid are exceptions to this rule.

Nausea and vomiting of early pregnancy, when associated with these vague abdominal pains or intestinal colic, may suggest the diagnosis of acute appendicitis, unless the menstrual history is elicited. The consequences are more serious, however, if the nausea, vomiting and pain caused by an acute appendicitis are attributed to an early pregnancy!

As a matter of convenience the pregnant period is divided into trimesters and various conditions which are encountered in each trimester follow.

The First Trimester.—A frequent complaint of early pregnancy is that of recurring sharp pains in both lower quadrants. They usually occur when the patient moves suddenly and are thought to be due to *tension on the round ligaments*. These pains tend to disappear when the enlarging uterus becomes more fixed.

Constipation is very troublesome during this time and may alternate with attacks of intestinal colic and diarrhea due to dietary indiscretions or purgation.

Abortion and ectopic pregnancy are most common in the first three months. Abortion of an intrauterine pregnancy begins with lower abdominal discomfort, regular uterine contractions develop, vaginal bleeding increases, and as the cervix dilates the products of conception are expelled, either completely or incompletely. When intrauterine or pelvic infection follows, the clinical pic-

ture is that of a pelvic peritonitis. Attempts to induce criminal abortion cause symptoms and signs which are most atypical. Hemorrhage and pelvic peritonitis are perhaps the most common complications. Ectopic pregnancy is the most frequently misdiagnosed gynecological disease! Bleeding from a corpus luteum of pregnancy simulates an ectopic pregnancy, but the acute signs and symptoms rapidly subside.

The pregnant uterus is often found to be retroflexed and retroverted in the first eight weeks but usually between the eighth and twelfth weeks spontaneous correction occurs. If the retroversion persists, impaction in the pelvis may result, and this may create very acute symptoms. Acute lower abdominal pain and retention of urine develop in a woman who considers herself pregnant. Overflow incontinence occurs. The large tender bladder is palpable and must not be mistaken for the enlarged uterus. On pelvic examination, the cervix is drawn up under the symphysis and the impacted, retroverted uterus fills the pelvic cavity. After the bladder is drained, it will be apparent on bimanual examination that the pelvic mass is the uterus. Its position can then be gently corrected if this does not occur spontaneously.

A pelvic hematocoele or an ovarian cyst always displaces the uterus, and therefore the latter is felt separate from the mass. A cervical fibroid produces pain, retention of urine, displacement of the cervix, but the symptoms and signs of pregnancy are not present.

Second Trimester.—In pregnancy, physiological distention and dilatation of the ureters cause stasis of urine, and acute pyelitis is a frequent complication. Right-sided pyelitis is more common. Ordinarily the symptoms, signs and urine analysis are diagnostic. Subacute renal infection may give rise to less typical symptoms and signs which are referable to the back or upper abdomen. This type of pyelitis may remain undiag-

nosed unless an acute exacerbation develops and a more complete renal tract investigation is made.

Renal calculi may cause attacks of colic for the first time during pregnancy. The symptoms and signs suggest the diagnosis and this is verified by routine investigation.

Acute appendicitis in pregnancy is a serious complication. An attack, very rarely the first, is most likely to develop in the first or second trimester, but may occur later. Nausea, vomiting, and abdominal discomfort, associated with the increased white cell count and rapid sedimentation rate, all normally present in pregnancy, tend to obscure the diagnosis. After the fifth month, the anatomical displacement of the cecum and appendix gives rise to atypical physical signs. An acute pyelitis of the right kidney must be excluded.

Once an acute appendicitis is diagnosed, operation is imperative. If there is peritonitis present, the chance of abortion or premature labor ensuing after operation is increased, and the maternal prognosis is more grave. It is important to note that peritonitis may cause uterine spasm which simulates the clinical signs of concealed retroplacental hemorrhage.

Perforation of a peptic ulcer during pregnancy has been reported. A diagnostic aid is the history of prior investigation for a suspected ulcer.

Intestinal obstruction due to adhesions from previous operations may occur as the adherent intestinal coils are displaced and kinked by the enlarging uterus. Paralytic ileus is a rare complication of acute pyelitis with pregnancy.

More commonly, acute abdominal pain in pregnancy is caused by *degeneration of a fibroid* or by an *ovarian cyst* which has undergone torsion, intracystic hemorrhage, or rupture. Such pelvic tumors are usually identified at antenatal examination, and the fact that their presence is already known aids in the diagnosis and treatment when complications develop.

A fibroid complicating pregnancy is ordinarily treated conservatively until the child is close to term. The treatment then is an obstetrical problem. An ovarian cyst associated with pregnancy should be removed by operation at a time when abortion is least likely to be precipitated, but before the bulk of the pregnant uterus makes the operation difficult.

A *hematoma in the rectus sheath* is a rare accident which occurs in the second trimester of pregnancy or later. The rupture of the muscle or blood vessel is usually caused by unusual muscular stress. The sharp pain, the tense abdominal wall with very acute local tenderness create a surgical emergency. The findings on abdominal palpation and pelvic examination while under the anesthetic clarify the diagnosis which is fully established when the hematoma is incised on opening the abdominal wall.

Third Trimester.—Again, in the third trimester, the patient may complain bitterly of discomfort due to the *pressure of the enlarged uterus*. This, of course, is aggravated if an acute hydramnios or a multiple pregnancy is present. Fetal movements may make certain points on the uterus very tender. Pain in the liver region in a patient with toxemia of pregnancy is a warning symptom of an impending eclampsia.

Retroplacental hemorrhage, if small, causes considerable distress which subsides in time. If the concealed hemorrhage is large, the distention of the uterus becomes marked, acute severe pain is present, and a state of shock ensues. There is usually an associated toxemia of pregnancy or a history of local trauma. On palpation the uterus is distended, hard, and tender. The fetal heart is absent.

Rupture of the pregnant uterus is a serious occurrence which usually happens near term or during labor. Spontaneous rupture is more common, and it is so frequent in cesarean section scars, whether in the upper or lower segment, that the dictum, "once a section always a section," is almost a rule.

An obstructed labor will end in rupture of the overdistended lower uterine segment if it is not recognized and treated. The unwise use of pituitrin in labor before the cervix is dilated may be the cause. Traumatic rupture may result from external trauma such as a blow on the abdominal wall, a gunshot wound, or a crushing injury. More frequently it is a direct result of forceful obstetrical procedures such as internal version late in labor. If the patient is in labor, the contractions cease, abdominal pain and tenderness develop. Shock with evidence of internal hemorrhage ensues, the fetal parts will be easily palpable, and the fetus usually dies. If the rupture occurs during delivery, the third stage is abnormal, there is delay in expression of the placenta, postpartum hemorrhage, and progressive shock. The diagnosis will not be made until one explores the uterine walls from within.

Occasionally an extrauterine pregnancy survives to the last trimester and abdominal pain and discomfort are distressing. The diagnosis is rarely made before the abdomen is opened because of the abdominal symptoms or for a proposed cesarean section.

The pain of abortion or the pain due to the onset of premature labor or labor at term are usually obvious and remain the most common causes of abdominal pain in women of the childbearing age, despite the misleading history sometimes given by the patient.

REFERENCES

- Brown, F. J.: *Antenatal and Postnatal Care*, ed 7, London, 1951, J and A Churchill, Ltd.
Curtis, A. H., and Huffman, J. W.: *Textbook of Gynecology*, ed 6, Philadelphia, 1950, W. B. Saunders Company.
Novak, E.: *Textbook of Gynecology*, ed 3, Baltimore, 1948, The Williams & Wilkins Company.
Shaw, Wilfred: *Textbook of Gynaecology*, ed 3, London, 1948, J & A Churchill, Ltd.

CHAPTER XXX

HERNIA

W. MASON COUPER, M.D.

Hernia may be defined as a protrusion of a viscus or a part of a viscus from the cavity in which it is normally contained through a congenital or acquired aperture or weakness. Subsequent descriptions will be restricted to types of abdominal hernias, e.g., inguinal, femoral, umbilical, and postoperative.

CONSIDERATION OF TERMS COMMON TO ALL HERNIAS

A *reducible hernia* refers to one in which the contents of the hernial sac can be replaced into the cavity from which they protruded, while an *irreducible hernia* is one in which the contents of the hernial sac cannot be so replaced.

An *incarcerated hernia* or obstructed hernia is one in which the hernial mass is irreducible. There is obstruction to the flow of intestinal contents in the occluded loop, but the blood supply and lymph drainage remain intact.

A *strangulated hernia* is one in which the blood supply and lymph drainage of the hernial contents are partially or completely occluded. This condition will result in gangrene unless relieved within a few hours.

TYPES OF HERNIA INGUINAL HERNIA

Embryological Features.—In the male, the close association of inguinal hernia with the descent of the testis necessitates a review of the embryological features and the path traveled by the testis in its descent. The testis develops as a retroperitoneal structure in the region of the kidney. During its descent it carries with it a tube of peritoneum through the inguinal canal and

into the scrotum. This protrusion of peritoneum is known as the *processus vaginalis*. Before birth, this processus becomes obliterated throughout its length, except for that portion which surrounds the testicle. This portion is known as the *tunica vaginalis*. When the processus vaginalis remains patent, there is a direct communication between the peritoneal cavity and the scrotum. The patent processus constitutes the potential sac for a future hernia.

Acquired and Congenital Factors Governing Inguinal Hernia.—In the male the majority of indirect inguinal hernias are considered to be congenital in origin. The peritoneum of a patent processus vaginalis has been found in 35% of infants up to the fourth month and many adults carry with them, throughout life, a patent funicular process. Factors such as coughing, straining, lifting in the adult and crying in the child, all tend to raise the intra-abdominal pressure and so put a strain on a partially closed, or a poorly obliterated processus vaginalis, with the development of a hernia. Unilateral or bilateral hernias are common in infants with phimosis, or pin-point meatus.

Classification of Inguinal Hernia.—There are two main types:

1. Oblique or indirect inguinal hernia.
2. Direct inguinal hernia.

There is a combination of the above types, which may fall into a third group, and is known as direct-indirect type of inguinal hernia. The protrusion is to both sides of the inferior epigastric artery.

Oblique or Indirect Inguinal Hernia

Oblique hernia is so designated because of the oblique course the hernia takes in

passing through the inguinal canal. The indirect hernia has been described according to the degree of patency of the funicular process. Where the process is open down to the testicle, the condition is described as the *congenital* or *testicular type*. When the dis-

obliterated, and a small intervening portion remains patent. This gives rise to a collection of fluid along the cord, cystic in nature, which is described as an *encysted hydrocele of the cord*. The cystic area may be uni- or multilocular.

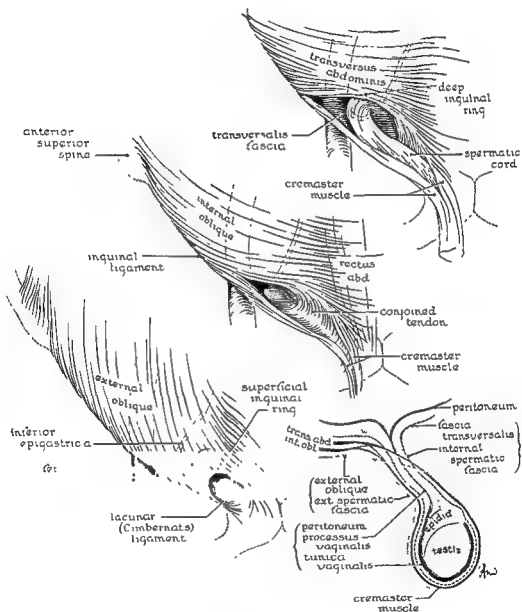


Fig 216—Anatomy of the inguinal canal

tal portion of the processus has been obliterated with patency of the proximal portion, it is described as the *funicular type*.

Again, there are cases in which the proximal and distal portions of the processus are

Oblique inguinal hernia occurs much less commonly in females than in males. The patent peritoneal diverticulum in the female corresponding to the processus vaginalis is called the canal of Nuck.

Coverings of Indirect Inguinal Hernia.—

The covering layers are:

1. The skin.
2. Superficial fasciae.
3. Intercolumnar fascia from external oblique aponeurosis

The separation of the coverings into layers will not be possible in the old, long-standing hernia, as the layers become fused together.

Contents of Sac.—Practically every abdominal organ, with the exception of the liver and pancreas, may, at one time or an-

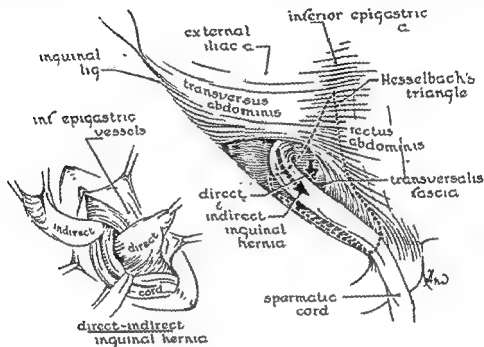


Fig 217—Types of inguinal hernia and anatomy of the posterior wall of the inguinal canal

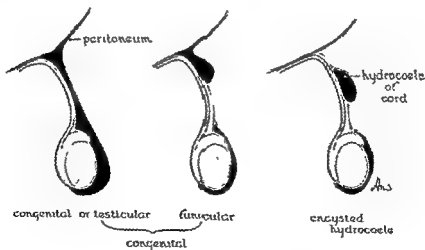


Fig. 218—Congenital anomalies of processus vaginalis

4. Cremasteric fascia.
5. The transversalis or infundibuliform fascia.
6. The extraperitoneal connective tissue.

other, descend into the hernial sac. When the sac in the oblique type contains loops of small bowel, the hernia is known as an *enterocele*. At times the omentum forms

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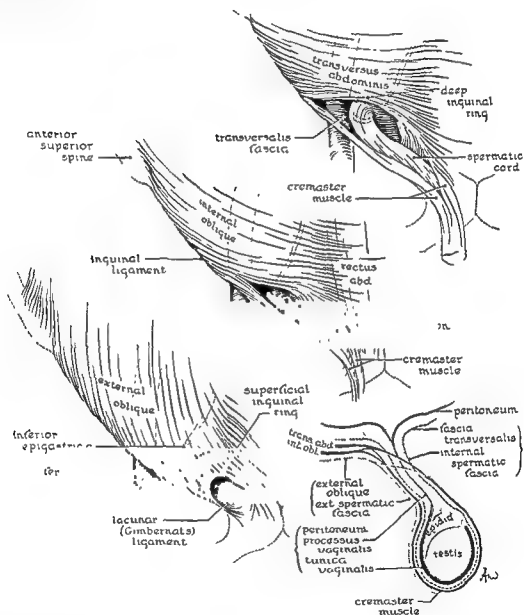


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relied upon as an important point in making the differentiation between these two types of hernia. More significant is the diffuse bulge passing directly forward from the peritoneal cavity characteristic of the direct type

Differential Diagnosis of Inguinal Hernia.—*Inguinal Adenitis.* A mass somewhat oval in shape presents in the groin. It is tender, painful and usually moves freely under the skin. Occasionally other lymphatic glands in the neighborhood, showing some enlargement and tenderness, are noted. The inguinal canal is found empty on examination and the enlarged gland or glands are palpated to one or other side of the canal

Ectopic testis, or a partially descended testis, may appear as an irreducible swelling in the inguinal region. This condition is readily recognized by the shape and consistency of the tumor, by the testicular sensation experienced by the patient when the swelling is compressed, and by the fact that no testis is present in the corresponding side of the scrotum.

Lipoma of cord consists of a soft lobulated fatty mass, not tender, not increasing greatly in size, and usually present for a considerable period of time.

Encysted hydrocoele of cord appears as a tense oval tumor, attached to the cord mass when the cord is made taut by traction on the testicle. It may be readily palpated in the external inguinal ring. The cyst can be transilluminated, which fact makes the diagnosis an easy one.

Psoas abscess may present just above Poupart's ligament or extend beyond it. The swelling is soft, fluctuant and may be diminished in size by compression. It is dull to percussion and gives a slight impulse on coughing. Clinical and radiological examination of the spine will disclose the source from which the abscess originated.

Hematoma of the Cord. A history of injury, with ecchymosis of the skin above

Poupart's ligament, with swelling along the cord usually enables one to make the diagnosis of a hematoma due to trauma.

Treatment—Indirect Inguinal Hernia.—Three methods have been used:

1. Mechanical by use of truss.
2. Surgical repair.
3. Fibrosis of the sac by injection of solutions.

Use of Truss.—This method has been used since ancient times, and the use of a type of bandage or belt has always been associated with the early references to hernia treatment. Not all inguinal hernias are suitable for operation, and although there are many difficulties attending the use of trusses, there are, nevertheless, indications. In the infant, especially up to the ages of 6 months to 2 years, a suitable truss may be worn and prove adequate in holding back the hernia until obliteration of the patent processus has been accomplished. A fairly large percentage of small inguinal hernias in infants do disappear under this method of treatment.

Adults are less likely to obtain a cure from the wearing of a truss but may derive considerable benefit by having well-fitted support. The adult should remove the truss after retiring and put it on before getting out of bed. It is preferable to wear the truss next to the body. Any underclothing beneath the pad does not facilitate proper fitting and encourages the hernia to slide down beneath or to one side of the truss. Should the hernia become irreducible, the truss cannot be worn. Trusses are contraindicated in cases of obesity, hydrocele, varicocele and incomplete descent of the testicle. For those patients with a chronic cough and the presence of a large hernia, it may be advisable to wear the truss at night. Irritation of the skin caused by the pad of the truss can be overcome by careful bathing and dusting the area liberally with talcum powder. Attention should be paid to the cleansing of the truss itself. When a truss is properly fitted, it should be worn without dis-

the only structure within the sac, and this condition is described as an *omentocele*. Occasionally a part of the wall of the small bowel is caught in such a way that only a portion of its lumen is occluded; this is known as *Richter's hernia*. When a Meckel's diverticulum is found in the hernial sac, it is known as *Littre's hernia*.

Clinical Features and Diagnosis.—At the onset, the characteristic feature is a dragging sensation in one or other groin. Following this pulling and dragging sensation a lump appears as a rounded swelling just above

When the patient is asked to cough, a swelling may be seen in one or other inguinal region. With the examining finger introduced into the inguinal canal (by invaginating the neck of the scrotum through the external ring), a definite impulse may be palpated after coughing, or at times, the examining finger is displaced beyond the external ring by the hernial protrusion. The course and obliquity of the hernia allow one to appreciate the indirect type. Symptoms of pain referred to the testicle of that side



Fig 219—Left complete (scrotal) inguinal hernia



Fig 220—Right complete (scrotal) inguinal hernia, operation demonstrated a hernia-en-glissade (sliding hernia)

Poupart's ligament and lateral to the spine of the pubis. Straining, coughing and running make the lump more obvious. On lying down, the swelling usually disappears. When the swelling emerges at the external ring, the condition is named an *incomplete inguinal hernia*, and when it descends beyond the external ring to gain the scrotum, it is designated a *complete* or *scrotal hernia*.

Examination of the patient is usually carried out with the examiner seated directly in front of the subject. The clothing is removed from the umbilicus to the knees

or gastrointestinal disturbances with occasional nausea are generally due to traction by a large hernia on the mesentery. Percussion of the hernia may enable the examiner to distinguish between intestine or fluid in the sac. A resonant note, on percussion, suggests gas in the intestines. The clinical differentiation between the oblique inguinal hernia and the direct type, by the palpation of the inferior epigastric artery, is extremely difficult and, therefore, cannot be

divided to expose the shiny aponeurosis of the external oblique muscle. The external ring should be isolated. At times the clear outline of the margins of the external ring cannot be seen, but the edges can be readily palpated by introducing the finger along the course of the cord where it emerges at the pubic spine; by following the cord upward, the prominent edges of the ring can be palpated. Now the aponeurosis is divided in the direction of its fibers downward through the external ring and upward to just beyond the internal ring. The ilioinguinal nerve will then be seen running along the cord lying on the cremasteric fibers. The cremasteric fibers are separated to expose the cord with its accompanying vessels. The sac in an indirect oblique hernia is found lying superficial to the cord. It is picked up and dissected from the surrounding structures. The neck is cleaned. The sac is opened and inspected for contents. The contents if viable are reduced. When strangulated non-viable bowel is found, it is resected. Adherent omentum is dissected free and returned to the abdominal cavity. To close the neck, a transfixion suture is used, or, if the neck be wider than two fingerbreadths, a purse-string suture may be used. The redundant part of the transfixed sac is excised. The second phase in the operation consists of a repair of the defect in the posterior wall of the canal. This is carried out by suturing the conjoined tendon down to the reflected edge of Poupart's ligament posterior to the cord. The external oblique is then closed over the cord and the superficial fasciae and skin sutured to complete the procedure.

Halsted added a modification to the above method by transposing the cord superficial to the sutured external oblique aponeurosis. In this way the posterior wall is given added strength by the use of the external oblique aponeurosis. In his earlier work, like Ferguson, he did not transpose the cord but sutured the conjoined tendon to Poupart's ligament, anterior to the cord except at the level

of the external ring where an opening was left for the passage of this structure.

In the treatment of an oblique hernia, in a very young infant or child, the procedure of high ligation of the sac is widely practiced. No attempt is made to disturb the contents of the inguinal canal. The sac is picked up at the external ring and freed at this point from the cord and vessels. Sufficient cleansing can be carried out at this level to permit the ligation of the sac and its excision close to the internal ring. Closure of Scarpa's fascia and the skin is all that is necessary.

Where a very large inguinal hernia exists the external and internal rings may come to lie against one another thus leaving a large defect in the posterior canal wall to be repaired. It is in this type that modifications of the above methods are used. The use of living fascia as strands to interlace the defect has been described by McArthur and Gallie. A large variety of materials has been used to overcome the defect—floss silk has been woven through the defect, also flagree wire and lately tantalum or stainless steel mesh or skin have all been used in selected cases.

Sliding Hernia or Hernia-en-Glissade

This type can be defined as a hernia in which the posterior wall of the sac consists of the cecum on the right side or the pelvic colon on the left side. It is found in the inguinal region and is brought about by the slipping, in a downward direction, of the viscus and the posterior parietal layer of peritoneum.

Symptoms and Diagnosis.—Sliding hernia of the large intestine is generally of the indirect inguinal type. It occurs in males over the age of forty-five years and is more common on the right side. If the hernia is reducible, the symptoms are very similar to those found in the ordinary inguinal hernia, with the exception that the sac is somewhat larger. The internal ring becomes stretched and lies over the external ring so that reduction of the hernia is usually easy, and strangulation seldom occurs. As the signs are not

comfort, and the patient should be able to stand up, bend down, cough, and do his work without protrusion of the hernia around the pad.

Surgical Treatment of Inguinal Hernia.—The fundamental principles which underly the surgical repair of inguinal hernia are thorough exploration of the canal, identification and excision of the peritoneal sac, and repair of the canal floor, or reconstruction of the defect in the abdominal wall.

the many modifications and methods by which a repair of the inguinal canal can be accomplished. Several well-recognized standard procedures will be used to illustrate the underlying principles.

The Bassini Operation embodies two main principles. removal of the hernial sac and repair of the defect in the posterior wall of the canal.

The operation is carried out by making an incision, parallel with Poupart's ligament and

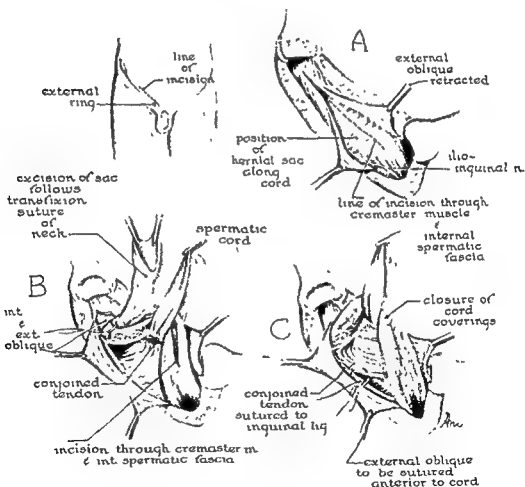


Fig. 221 —Bassini repair of indirect inguinal hernia.

The methods of carrying out the surgical repair are many and varied. Indeed, practically every known procedure has been either rediscovered, modified or rewritten in an effort to seek the perfect repair. No attempt will be made to review or describe

one-half inch above it, extending from the spine of the pubis to beyond the internal ring (one-half inch above the midpoint of Poupart's ligament). Camper's fascia, represented by the thick subcutaneous yellow fat, as well as the deeper Scarpa's fascia, are

need not be excised but may be pushed back through the defect in the transversalis fascia by invagination, with repair of the fascial defect. The conjoint tendon is sutured to Poupart's ligament over the invaginated sac beneath the cord. When a larger sac is present, it should be opened and the redundant tissue excised. The peritoneal defect is then closed. The remaining features in the repair are as described previously.

The combined direct-indirect inguinal hernia is converted into a true oblique type and dealt with as such.

Complications Following Operative Repair of Hernia.—Careful preoperative care and thorough cleansing of the area before operation do a great deal to lessen the incidence of wound infection. The introduction of antibiotics has further helped to reduce this problem and the practice of giving an antibiotic routinely is used by many surgeons. The use of nonabsorbable suture material (silk, cotton or wire) results in less postoperative tissue reaction and a lower recurrence rate. Occasionally sinuses may supervene if infection occurs from the presence of this foreign material.

Hematoma is one of the more frequent complications of *herniotomy*. It is most common after the removal of the large scrotal sac. The hematoma may be confined to the cord, to the inguinal canal or subcutaneous tissues or may be large and fill the scrotum. If it is extensive, the wound should be reopened at the earliest moment and the blood evacuated. Chemotherapy should be routinely used to prevent secondary infection.

Lung complications, mostly due to atelectasis, may develop. It has been found that these complications occur least with general anesthesia and somewhat more frequently with spinal and local infiltration. Pulmonary embolism has occurred.

The later complication of recurrence of the hernia varies a great deal from clinic to clinic, the percentage of recurrences ranges from 5 to 20%. The mortality rate is low

for this operation being under 1%. This, of course, does not include the cases of inguinal hernia complicated by bowel obstruction and strangulation.

FEMORAL HERNIA

A femoral hernia is one in which the hernial protrusion takes place through the femoral ring into the femoral canal.

The boundaries of the femoral ring are medially the lacunar (Gimbernat's) ligament, laterally the femoral vein, posteriorly the ramus of the pubis, covered by the pectineus fascia and ligament, and anteriorly the inguinal ligament.

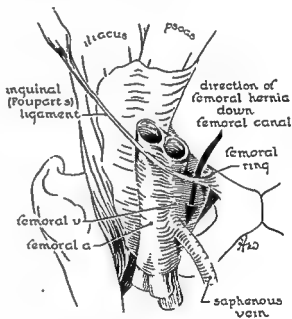


Fig. 223 —Anatomy of femoral canal.

Etiology, Sex, and Incidence.—Femoral hernia is found much more frequently in females than in males, in the ratio of 3 to 1. The condition is rarely found in children. When present in adults it may be unilateral or bilateral.

The etiology of the development of femoral hernia, on a congenital basis, is not so universally accepted as in the case of indirect inguinal hernia. There are many who feel there is, in this type of hernia, a pre-formed pouch or peritoneal diverticulum

well defined in this type of hernia, the diagnosis is not made until operation. On the left side, the presence of the colon in the sac may give rise to symptoms of constipation. The diagnosis may, at times, be established by radiological examination of the colon, the barium being seen in the hernial sac. Intestinal obstruction, following such a procedure, can occur with the barium becoming inspissated in the herniated colon.

Treatment of Sliding Hernia.—The use of a truss is not satisfactory in this type of hernia because of the large size of the hernial opening. Radical operation is the treatment of choice. The exposure of the sac is the same as in the ordinary type of inguinal hernia. In this type, the bowel forms the major portion of the posterior wall of the sac. Accidental opening of the colon can easily occur while exposing the sac. The redundant portion of peritoneum is cut away and the colon mobilized to allow it to be returned to the abdomen. The repair of the defect is difficult. Many writers suggest opening the abdominal cavity and fixing the colon to the posterior abdominal wall. Others complete the repair by bringing the conjoined tendon down to the reflected edge of Poupart's ligament, as in the ordinary oblique inguinal hernia.

Recurrence is very common following the repair of this type of hernia because of the extensive defect and the tendency of the viscus to slip down again.

Direct Inguinal Hernia

A direct inguinal hernia is one in which the protrusion takes place through Hesselbach's triangle. This triangle is bounded medially by the lateral border of the rectus abdominis, below by Poupart's ligament, and laterally by the inferior or deep epigastric artery. The hernia is rarely if ever on a congenital basis. It may be unilateral or bilateral. It is almost entirely confined to males after the fourth decade. It is thought to be due to weakness in the transversalis fascia with atrophy of the conjoined tendon.

Clinical Features and Diagnosis.—There is an oval swelling in the groin, which presents above Poupart's ligament. The swelling increases in size on coughing and is reducible, disappears on lying down. On examination, the finger, when inserted into the external inguinal ring, gives the examiner the sensation of going directly back into the abdomen, whereas in the indirect type, the



Fig 222.—Direct inguinal hernia

finger takes an upward course along the inguinal canal. The swelling rarely descends into the scrotum. There is a dragging sensation but less pain than in the indirect type. At times it is impossible to differentiate between the two types; the final diagnosis is made at operation. Palpation of the deep epigastric artery to distinguish between the indirect and direct types is seldom possible, and rarely can be relied upon in establishing the diagnosis. The differential features are those described in the oblique type.

Treatment in Direct Inguinal Hernia.—In selected cases, conservative methods, such as the wearing of a truss, may suffice, but the majority of these cases come to operation. The operative features follow the pattern described under indirect hernia, except for the handling of the sac. When small it

are saphenous varix, incomplete inguinal hernia, psoas abscess, and regional lymphadenopathy.

The *saphenous varix* disappears when the patient lies down and can be readily compressed by gentle pressure over that site. There is no gurgling and occasionally a venous hum is heard on auscultation over the area. A venous thrill may be felt on coughing. The skin may show a bluish discoloration over the saphenous varix, and there may be varicose veins on that extremity.

Progressive enlargement of a femoral hernia occurs by protrusion through the fossa ovalis, the sac being turned upward by the fascial arrangements, and the differentiation between it and an *incomplete reducible inguinal hernia* is at times difficult. Here, the inguinal hernia can be reduced by pressure exerted in an upward, outward, and backward direction, and continued pressure over the inguinal canal will prevent its descent. The reduction is much quicker than in femoral hernia where the necessary pressure is usually downward and medially. The inguinal hernia will be seen to lie above a line running from the spine of the pubis to the anterior superior spine, whereas the femoral hernia is found below such a line.

Psoas abscess. When such an abscess presents below Poupart's ligament, it is a soft fluctuant mass transmitting an impulse on coughing. It is reducible in some cases without any gurgling sound or sensation. Fluctuation may be present in a dumbbell-shaped abscess above and below Poupart's ligament. Examination of the spine usually reveals other evidence of tuberculosis.

Lymphadenopathy in the femoral region may give rise to difficulty in diagnosis. Here the swelling is firmer, with tenderness on pressure; usually there are other enlarged glands in the neighborhood and a search may reveal some focus of infection about the leg or foot to give rise to the adenopathy. In certain cases, the diagnosis may be established only at operation.

Treatment.—The cure of femoral hernia is by operation. The tendency for this type of hernia to strangulate indicates early surgical intervention. Mechanical treatment is unsatisfactory and offers little chance of cure; however, there are cases in which for some medical reason, operation is inadvisable and a truss may be worn. The site of this hernia makes the wearing of the truss difficult, as the pressure pad can be readily displaced by the movements of the body and thigh. For these reasons the operative treatment is recommended. The operation is directed toward removal of the sac and closure of the femoral ring.

Operations for Femoral Hernia.—There are two recognized approaches for the surgical repair:

1. The inguinal route.
2. The femoral route.

Each method has its advocates and a brief description of each will be given.

Repair by the Inguinal Route.—This procedure was first described by Annandale and continues to retain that name. The incision is made $\frac{1}{2}$ inch above Poupart's ligament and parallel with it for a distance of 3 or 4 inches. The aponeurosis of the external oblique muscle is divided in the direction of its fibers over the inguinal canal. By retracting the edges of the divided aponeurosis of the external oblique, the conjoined tendon, spermatic cord, and transversalis are brought into view. Gentle retraction of the cord upward exposes the floor of the inguinal canal, which consists of the transversalis fascia. An incision is then made in the transversalis fascia along the line of the inguinal canal and over the neck of the sac. This exposes the peritoneum, which is traced to the neck of the sac, and the sac, if small, can be freed and pulled back, or reduced. The fundus of the sac is picked up in forceps, opened, and tags of adherent omentum are freed. The neck is transfixed as in the ordinary inguinal hernia operation or closed with a purse-string suture. The redundant portion of the sac is removed, and the transfixed portion is

which has failed to become obliterated. This fact is borne out by the finding on many postmortem examinations of a preformed peritoneal sac in the femoral canal. A great many femoral hernias, especially in the older age group, are thought to be acquired. In the older age group, the existence of lax muscles, attended by loss of weight and poor tissue tone, seems to predispose to the development of this type of hernia.

but never completely disappear. There remains a small fatty nodule after the main contents of the sac have been reduced. This remaining nodule may represent the fatty plug filling in the femoral ring along with the large lymph node at the apex of the femoral canal (gland of Cloquet). Pain is not a prominent feature; but if omentum or intestine is contained in the sac, the patient may complain of a dragging sensation or colicky pain. The pain usually radiates



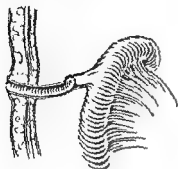
Fig 224—Femoral hernias

Clinical Features and Diagnosis.—The symptoms are much less marked in femoral than in inguinal hernia because the hernia is small and if no intestine is contained in the sac, it may be overlooked altogether in obese patients. The hernia appears as a small globular swelling just lateral to the spine of the pubis and below Poupart's ligament. Unless the sac contains intestine, there may be very little increase in size on coughing, and when the patient lies down the contents of the sac may reduce slightly

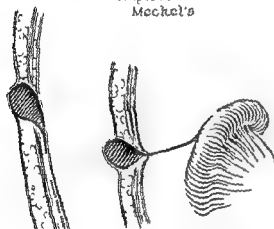
down the anteromedial aspect of the thigh. Gurgling may be heard and felt if intestine is present in the sac. Strangulation in this type of hernia is more common than in the inguinal variety because of the small size of the ring and the rigidity of the surrounding structure. Richter's hernia, in which only a part of the lumen is caught by the femoral ring, is more likely to occur in femoral than in any other variety of hernia.

Differential Diagnosis.—Those conditions most commonly mistaken for femoral hernia

few symptoms evinced by the patient. The diagnosis is never a difficult one as the presence of the bulge in the region of the umbilicus along with the small opening in the ring makes recognition easy.

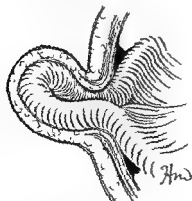


complete
Meckel's



umbilical cyst
of Meckel's

bladder
cyst of urachus
at umbilicus



umbilical hernia

Treatment.—It is in the infantile type that mechanical aids in the treatment of hernia play an important part and the wearing of a pad and adhesive strapping to cover the umbilical opening has resulted in a great many cures. In this method of treatment, care must be exercised to see that the pad is larger than the defect in the ring otherwise it will act as a cork in a bottle and prevent scar formation from closing the ring. The strapping should be applied so as to cause infolding of the umbilical area. When the treatment is begun, usually at the age of one month, the strapping must be continued without interruption for a period of 3 to 6 months. It is necessary to change the bandage every 3 to 4 weeks.

There are cases, usually in patients older than 18 to 24 months, in which this type of strapping will not suffice to hold back the hernia. It is in these cases that a more radical approach is necessary to obtain a cure. Operative treatment is indicated when the hernia is large or when attacks of abdominal pain with colic and vomiting occur, indicating a partial intestinal obstruction. The umbilical ring in these cases can admit the index finger quite readily. Two procedures are employed. The first is described as the *subcutaneous ligature of the sac neck*. The use of this method depends on the size of the umbilical opening and the fact that the contents of the sac can be reduced. After reduction of its contents, the fundus of the sac, that is, the umbilicus, is grasped in forceps and lifted upward. Three small incisions are made radiating from the umbilicus equidistant around the periphery of the sac neck. These incisions go down to the sac wall. By means of a blunt instrument (artery forceps), the three incisions are connected by a subcutaneous tunnel around the navel. Through this tunnel the ligature is passed, both ends emerging at the site of entry. After careful palpation of the sac to determine the reduction of its contents, the ligature is tied

allowed to drop back into the abdomen. The opening in the femoral ring is now closed with interrupted sutures. These run from the lower margin of Poupart's ligament to the ligamentous reflection along the ramus of the pubis (Cooper's ligament), occasionally to the pectineal fascia. Three or four sutures usually suffice to close the opening. The conjoined tendon is then brought down to Poupart's ligament, beneath the cord. The external oblique and skin are closed in the usual manner.

In the Lotheisen repair, the conjoined tendon is sutured behind the cord to Cooper's ligament.

Repair by the Femoral Route.—The incision is made below and parallel with Poupart's ligament over the prominent part of the swelling. The soft tissues and fat are separated to expose the sac lying embedded in the fat of the thigh. The sac is dissected free to expose the femoral ring from below, care being exercised not to damage the femoral vein lying lateral to it. The constriction about the neck is stretched to enlarge the opening. The sac is opened with care to avoid the possibility of injuring the contents. If the sac contains omentum or intestine, this is reduced and the neck of the sac is pulled down, closed, in the method described above, and the redundant portion is removed, allowing the transfixed neck to slide back through the femoral ring behind Poupart's ligament. The femoral ring is then closed by bringing the lower margin of Poupart's ligament down to the pectineal fascia.

Strangulated Femoral Hernia

Here, the inguinal approach is preferable. The peritoneum, when exposed, can be opened to identify the ring and the obstructed intestine. If the intestine is not viable, a resection is much easier to carry out than by the femoral approach. There are cases where the constricting ring cannot be overcome by this approach, and a combined inguinal and femoral approach

may be necessary in order to free the adherent mass in the femoral region. At times the constricting ring may not yield, and it may be necessary to incise Poupart's ligament to free the constriction and expose the area.

UMBILICAL HERNIA

An umbilical hernia is one in which the protrusion takes place through the umbilical ring. The majority of such hernias are congenital in origin and appear in children. The condition is caused by failure of the umbilical ring to close following the section of the umbilical cord. This type of hernia may also be acquired and appears later in life in adults.

The discussion of umbilical hernia will include what is often described as paraumbilical hernia. This type is characterized by protrusions through small fascial defects adjacent to the umbilical ring and comprises a large group of umbilical protrusions occurring in the aged or obese female.

Umbilical Hernia in Infants

The peritoneal process in the umbilical cord is usually obliterated at birth. Sometimes it persists and gives rise to a congenital hernia containing intestine and the unobliterated omphalomesenteric duct. This condition is called *exomphalos*.

The usual type of infantile umbilical hernia develops in the first six months of life and is thought to be brought about by a weakening in the scar tissue closing the umbilical ring. This weakness is caused by factors which increase intra-abdominal pressure such as straining or crying. The condition is recognized by the small, round bulge appearing at the umbilicus and made more prominent by crying. The small defect in the umbilical ring may be felt by introducing the tip of the little finger into the opening. The size of the protrusion may vary from that of a small marble to the size of a fist. The sac of the infantile hernia rarely contains bowel. This fact accounts for the

patient should be prepared for operation by suitable dietary regime and re-education of the abdominal musculature.

The method of choice is that described by Mayo. This consists of making a large elliptical incision in a transverse direction about the umbilical mass. This usually includes a large area of the adjacent fat. The incision is deepened to expose the aponeurosis on both sides of the umbilicus. The hernial mass and fat are dissected toward the umbilical ring until the neck of the sac is exposed throughout its circumference.

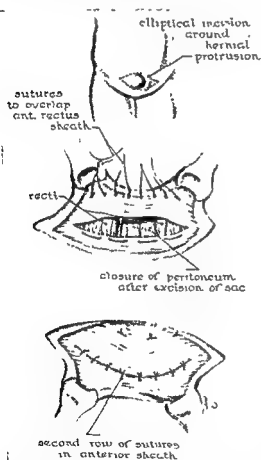


Fig. 227—Mayo operation for repair of umbilical hernia

The sac is opened near the neck, the adhesions are separated, and the hernial contents are reduced into the abdominal cavity. To facilitate reduction, the hernial ring is enlarged and a portion of adherent

omentum is resected. The excess margin of the peritoneal sac is cut away and the edges picked up in forceps. To close the umbilical ring, the aponeurosis is incised in a transverse direction. It is usually impossible, in the large hernia, to close the peritoneum as a separate layer. The fascial edges with the peritoneum are picked up and overlapped, chromic catgut or heavy silk sutures being used. The free margin of the overlapped edges is sewn down with interrupted or continuous sutures. The thick layer of skin and subcutaneous tissue is then closed, care being taken to avoid any dead space. A dressing is applied and held in place by adhesive straps or a scultetus bandage.

VENTRAL HERNIA

Any protrusion through the anterior abdominal wall, other than at the inguinal, umbilical or femoral region, is defined as a ventral hernia.

Ventral hernias may be classified into three groups:

1. Epigastric hernia, or hernia in the linea alba.
2. Hernia in the linea semilunaris.
3. Incisional or postoperative.

Epigastric Hernia, or Hernia in the Linea Alba

Epigastric hernia is a protrusion of the abdominal viscera through an opening in the linea alba. It occurs above the umbilicus and is found rarely in children. The actual protrusion is preperitoneal fat which forces its way through the fascial interstices of the linea alba, at times following the perforating arteries. Once the fatty protrusion has started, intra-abdominal pressure augments its size until a real sac of peritoneum is finally pushed out from the abdominal cavity.

Symptoms and Diagnosis.—The condition may be present and give rise to no symptoms whatsoever and be found accidentally on examination. A small, soft tumor, at

tightly. The three small incisions are each closed with one or two fine sutures.

A second method for radical operation is the same as that carried out in older patients and consists of reconstructing the existing defect of the umbilical ring. This procedure is referred to as the Mayo repair. The details of this method will be described below under the treatment of adult umbilical hernia.

Umbilical Hernia in the Adult

In the older patient, this type of hernia is usually acquired. It appears between the ages of 30 and 55 years. The etiological factors are increased intra-abdominal pressure, relaxation of the abdominal muscles,



Fig 226—Umbilical hernia in obese patient with relaxed abdominal musculature.

obesity, ascites, multiple pregnancies and diastasis recti. The umbilical opening, being a weak point in the abdominal wall, gives way and the protrusion develops.

Women are more prone than men to develop this type of hernia. The size of the hernia varies and may reach considerable proportions.

Symptomatology.—In the early case, in the obese patient, a small hernia may not be visible and is found on palpation as a rounded, firm nodule in the region of the umbilicus.

In the early stages an expansile impulse is seen when the patient coughs or strains; the tumor if reducible disappears when the patient lies down. At times the hernia appears to emerge at one or other side of the ring and for this reason the term paraumbilical hernia has been introduced. When the hernia increases in size, it presents as an oval or rounded mass, which as it enlarges becomes pendulous and may reach considerable proportions. The sac becomes lobulated and irregular in outline. The contents are usually the omentum, along with the transverse colon and occasionally the small intestine. Because of the tendency of the omentum to become adherent to the walls of the sac, this type of hernia rapidly becomes irreducible. When bowel is included, peristalsis may be observed.

The patient complains of pain usually dragging in character. Traction on the colon gives rise to attacks of colicky pain with constipation, and at times, nausea and vomiting, especially when strangulation develops.

Treatment.—The treatment is difficult as the majority of these patients are in the older age group with poor musculature and are usually obese. The small hernia, if reducible, may be controlled by a truss. In the obese subject an elastic belt may be worn. In the large hernia, operative repair is necessary except in those cases in which there are contraindications, such as cardiac or pulmonary disease. In some patients with very large protrusions, repair is inadvisable because of the danger of increasing intra-abdominal pressure which may produce severe pulmonary embarrassment. The

down over the abdomen as in the large umbilical variety. The diagnosis is not a difficult one, with the history of an operation and the development of a slowly enlarging protrusion through that incision.

Treatment.—In certain cases considerable relief may be obtained by a properly fitted abdominal support. Curative treatment is by operation if the general condition of the patient permits. The operative procedure follows that described for umbilical hernia. The old scar is excised, the contents of the sac are reduced, and the repair of the defect is carried out by overlapping the edges. When this is not feasible, some form of patch to fill in the defect may be used. Skin, fascia, tantalum, and stainless steel mesh have been recommended.

DIASTASIS OF THE RECTUS MUSCLES

Diastasis of the rectus muscles is not a true hernia, it is brought about by stretching or widening of the linea alba in such a way as to separate the recti muscles. Two distinct types are found: infantile and adult.

Diastasis in the Infant

The condition is noted shortly after birth when a large oblong bulge is seen. It runs from the xiphoid to the umbilicus, and is made more prominent when the child cries or strains. The child is usually undernourished or premature. The gap can be readily felt by palpation along the linea alba. No symptoms are present and no active treatment is necessary. The condition disappears as the child develops.

Diastasis in the Adult

Separation of the recti is found most frequently in the middle-aged female, who has had numerous pregnancies. The diastasis is made more prominent by raising the head and shoulders from the bed. The large gap can be easily felt with the hand placed along the midline as the hand sinks readily

into the abdominal cavity. A large number of these cases are asymptomatic, but there are some who complain of a weak feeling and dragging sensation in the lower abdomen.

The treatment is usually supportive or mechanical, with the aid of a properly fitting abdominal belt, but when this does not relieve the condition, operation may be necessary. It is carried out along the lines described above under ventral hernia.

RARER TYPES OF HERNIA

This group includes a number of unusual hernias which are designated by the names of the anatomical sites from which the protrusions occur; e.g., obturator, sciatic, lumbar, or perineal hernia.

Obturator Hernia

An obturator hernia is one in which the protrusion passes through the obturator foramen. The hernia leaves the pelvis following the course of the obturator vessels and nerve, to enter the thigh at the upper and inner aspect. The relationship of the obturator vessels and nerve to the sac varies. At times the sac may be to one or other side of the vessels and occasionally it divides and is found on both sides of the vessels. This hernia is found mostly in older women and in the emaciated patient.

Symptomatology and Diagnosis.—The symptoms may be intermittent with pain along the inner side of the thigh. There is a characteristic distribution of the pain which follows the course of the obturator nerve. The pain, because of its distribution and character, has been described as the Howship-Romberg sign. When present it is pathognomonic of the condition. The neck of the sac can sometimes be felt on vaginal or rectal examination. However, a tumor can rarely be palpated on external examination except in the case of a very large hernia. Gastrointestinal symptoms when present are usually nausea, colicky pain, vomiting, and constipation. This

times no larger than the tip of one's finger, may be palpated in the midline between the xiphoid and the umbilicus. The tumor may be present just to the right or left of the midline. There may be localized tenderness. When major symptoms are attributable to this hernia, a true peritoneal sac is usually present which may contain local viscera. In lesser degrees of such herniation where symptoms are present, a careful investigation of the gastrointestinal tract should be carried out before correction of the hernia. At times, symptoms of gastric distress, nausea, belching, vomiting, and a dragging sensation in the pit of the stomach may be present. The condition may be aggravated by bending over or sneezing. When the patient lies down the small tumor mass may be reduced with relief of the symptoms. A small fibrous ring is palpable at times with the tip of the finger. In a large number of cases reduction cannot be carried out and pressure on the protrusion simply mushrooms it over the fibrous opening.

Treatment.—There is little place for mechanical treatment in this type of hernia. Operative correction is carried out by making a longitudinal or transverse incision over the hernia exposing the fatty tumor of preperitoneal fat, and if small, removing it after ligating the small artery running alongside. The defect in the fascia is closed with one or two interrupted sutures. In the larger protrusions, when a tent of peritoneum is present in the ring, the neck of the sac is ligated as described for umbilical hernia, and the fascia is closed.

Hernia in the Linea Semilunaris

The semilunar line runs from the cartilage of the 9th rib to the pubic spine and corresponds roughly to the lateral border of the rectus abdominis muscle on each side. A protrusion through this line has been called *Spiegel's hernia*. This hernia generally occurs in middle life; the sex incidence is about equal. The condition is generally acquired, though some writers refer to a traumatic

variety. The vast majority develop slowly and spontaneously.

The hernia is commonly small and seldom reaches in diameter more than one inch. The symptomatology is not characteristic. The presence of a small lump below the umbilicus at the lateral border of the rectus muscle, which in most instances disappears when the patient lies down, is diagnostic. The treatment is surgical following the method outlined for the epigastric hernia.

Incisional or Postoperative Hernia

It is in this group that the largest number of ventral hernias occur. These hernias occur through previous operative sites on the anterior abdominal wall.

Certain etiologic factors play a part in the development of this type of hernia.

1. Postoperative infection in a wound, especially when drainage was required.

2. Improper closure of the original wound allowing the omentum to escape between the sutures.

3. Improper suture material

Prophylactically, the incision should be placed to avoid unnecessary damage to motor nerves, muscles, fasciae, and blood vessels.

Symptoms and Diagnosis.—The patient complains of a weakness and a bulge in the region of a previous operative scar. The symptoms are similar to those of umbilical hernia, and are aggravated by exercise and coughing. The protruded mass may not reduce on lying down, and complete reduction in the larger hernia is usually impossible, since the omentum becomes adherent to the walls of the sac as in the large umbilical hernia. In the incisional hernia, the protruded mass spreads out under the subcutaneous fat tissues in such a way that strangulation is not uncommon and when present manifests the symptoms of an acute intestinal obstruction with crampy pain, vomiting, and distention. The hernia may attain a considerable size and tend to hang

REFERENCES

- Annandale, Thomas: Case in Which a Reducible Oblique and Direct Inguinal and Femoral Hernia Existed on the Same Side, and Were Successfully Treated by Operation, *Edinburgh M. J.* 21: 1087, 1875-1876.
- Bassini, E.: Ueber die Behandlung des Leistenbruchs, *Arch. f. klin. Chir.* 40: 429, 1890.
- Gallie, W. E., and LeMesurier, A. B.: Living Sutures in Operative Surgery, *Canad. M. A. J.* 11: 504, 1921.
- Halstead, W. S.: The Radical Cure of Inguinal Hernia in the Male. *Johns Hopkins Hosp. Bull.* 4: 17-24, 1893.
- Koontz, A. R.: Preliminary Report on Use of Tantalum Mesh in Repair of Ventral Hernias, *Ann. Surg.* 127: 1079-85, 1948.
- Lotheissen, G.: Zur Operation der Schenkel Hernie, 2nd, *Congrès de la société internationale de chirurgie, Bruxelles*, 1908, vol. 1, p. 399.
- Maingot, R.: Floss Silk Lattice Posterior Repair Operation for Direct Inguinal Hernia, *Brit. M. J.* 1: 777-778, 1941.
- Mair, G. B.: Analysis of Series of 454 Inguinal Herniae With Special Reference to Morbidity and Recurrence After Whole Skin-graft Method, *Brit. J. Surg.* 34: 42-48, 1946.
- Mair, G. B.: Preliminary Report on Use of Whole Skin Grafts as Substitute for Fascial Sutures on Treatment of Herniae, *Brit. J. Surg.* 32: 381-385, 1945.
- McArthur, L. L.: Autoplastic Suture in Hernia, and Other Diastases. Preliminary Report, *J. A. M. A.* 37: 1162, 1901.
- McVay, Chester B., and Anson, B. J.: Inguinal and Femoral Hernioplasty, *Surg., Gynec. & Obst.* 88: 473, 1949.
- Rehn, E.: Das kutane und subkutane Bindegewebe als plastisches Material, *München. med. Wchnschr.* 61: 118-121, 1914.
- Watson, Leigh, F.: Hernia: Anatomy, Etiology, Symptoms, Diagnosis, Differential Diagnosis, Prognosis, and the Operative and Injection Treatment, ed. 2, St Louis, 1938, The C. V. Mosby Company.
- Wernicke, H. O.: Injection Treatment of Hernia, *Surg., Gynec. & Obst.* 68: 1093-1098, 1939.

hernia is rarely diagnosed before complications arise, that is, before intestinal strangulation occurs. The leg on the side of the hernia may assume an attitude of flexion, internal rotation, and adduction to relax the associated muscles. Coughing sometimes accentuates the pain in the thigh.

Treatment.—The treatment is operative and the best approach is by the abdominal route. This has the advantage over the obturator approach of not requiring another incision when the diagnosis has been made, and carries less risk of damage to the obturator artery and nerve. By opening the abdomen with a lower abdominal incision, the obstructed intestine is easily found and examined. The portion of obstructed bowel may be replaced by gentle traction from within, aided by pressure exerted from without over the thigh. If resection is necessary, it can be carried out readily. The sac is inverted and the base transfixed. The redundant portion is cut away and the defect closed by bringing together the adjacent tissues.

Lumbar Hernia or Petit's Hernia

In this hernia the protrusion takes place in the lumbar region, in the space between the 12th rib and the crest of the ilium and through Petit's triangle. There are two types:

1. The congenital, which develops spontaneously as a diffuse bulge in this area

2. The acquired, which occurs in adults and usually follows trauma or operations in this area

The soft mass is easily reducible, and strangulation seldom occurs. Diagnosis is easy when a bulge develops in this location which presents an impulse on coughing and a resonant note on percussion.

Treatment.—This is usually conservative with the aid of a supporting abdominal belt. There are, however, certain cases in which the abdominal symptoms of dragging pain, nausea and vomiting necessitate an operative repair.

Perineal Hernia

Perineal hernia is one in which the protrusion takes place through the muscles and fasciae of the pelvic outlet. It is most common in women between the ages of 40 and 60 years. It can occur in males where the protrusion is into the ischiorectal space. There is no definite symptomatology; the occurrence of a soft swelling in the region of the labia and ischiorectal fossae should include this condition in the differential diagnosis.

Treatment.—The defect may be repaired by an abdominal route or at times by a combined perineal and abdominal approach.

Maydl's Strangulated Hernia

Maydl's strangulated hernia is not really a condition to be described as a rare hernia but represents a retrograde strangulation of intestine in an inguinal hernia. The condition is brought about by a loop of small intestine passing into a sac and then back again into the abdomen, in such a manner as to resemble the letter "W." All the loops may be strangulated or only the loop returning into the abdomen may be obstructed. The signs and symptoms are those of a strangulated irreducible inguinal hernia and the treatment follows that for the relief of strangulation.

INJECTION TREATMENT OF HERNIA

It was thought possible to cure hernia by the injection of a sclerosing solution into and around the neck of the sac, and successful obliteration of the sac has been reported in selected cases, particularly of the indirect inguinal type. The complications, however, which may follow the injection of sclerosing fluids in the vicinity of delicate structures, such as the spermatic cord, have diminished the popularity of this method of treatment.

If the facts do not appear in the history, note the absence of:

- (a) Hematuria.
- (b) Passing of sand, gravel, or calculus.
- (c) Chill.
- (d) Fever.

Physical Examination:

- (a) General.
- (b) Local—genitourinary system.

Kidneys:

On examination note:

1. Tenderness in costolumbar angle.
2. Kidneys palpable (bimanual examination).
3. Tenderness of kidney to palpation.
4. Mass in renal region—description of size, extent and mobility; ballotable or movable into loin. Describe what you see, feel, and perceive. Note any tenderness, rigidity, or splinting of the muscles of the anterior abdominal wall in the renal region.

Ureters:

Examine for tenderness along course of ureters

Describe site and extent of tenderness and any masses palpated.

Bladder and Urethra:

Inspect suprapubic area; describe visible masses as to size, shape and extent, and by palpation any tenderness present, the consistency of any mass and whether it is indurated or not. Define the exact area of the mass by percussion.

Urethral Meatus:

Inspect meatus for size, discharge (smear), evidence of acute or chronic inflammation, indurations (meatal chancre).

Examine by external palpation the course of the urethra from the meatus to its disappearance in the perineum. Note indurations and fistulas

Explore the urethra with soft rubber catheter (F. No. 14).

Note any obstruction found, the site, and whether it can be passed.

Use gentleness at all times so as to avoid making a false passage.

Record residual urine and bladder capacity in cubic centimeters or ounces.

Penis:

Examine for scars, evidence of inflammation, rash.

Scrotum:

Inspect for evidence of inflammation, induration or eruptions. Describe any masses felt, their consistency, transmission of light, impulse on coughing and whether they extend into the inguinal canal.

Testes:

Describe size, consistency, induration, and whether any inflammation is present. Note if testicular sensation is normal.

Epididymes:

Describe size, consistency, tenderness, indurations.

Vasa:

Describe indurations, nodules, tenderness.

Veins of Pampiniform Plexus:

Varicocele; note if any present.

Rectal Examination:

- (a) Note if sphincter tone is normal.
- (b) Describe prostate as to size, shape, consistency, nodules, fluctuation, and fixation to surrounding tissues.
- (c) Carcinoma of rectum—note whether present or absent.

Seminal Vesicles:

Palpate for induration, nodules or tenderness.

Laboratory Work

Routine on admission:

1. Routine specimen of urine, including gross appearance, specific gravity; pH; sugar; albumin and microscopic examination.
2. P.S.P. renal function.
3. Wassermann.
4. N.P.N.

CHAPTER XXXI

GENITOURINARY SYSTEM

JOHN T. MACLEAN, M.D.

GENITOURINARY SURGERY

In genitourinary surgery it is essential that fundamental facts be recognized as such. These facts, combined with experience, provide a basic concept of the subject. It is for this reason that the various facts will be presented under seven main divisions, and these in turn will be applied to each area, e.g., kidney, bladder, etc. These main divisions are:

1. Anatomy and embryology
2. Normal physiology
3. Congenital abnormalities
4. Infections.
5. Tumors
6. Trauma
7. Foreign bodies

Pyogenic infections, tuberculosis, and calculi will be discussed separately

A carefully elicited history, physical examination, and urine analysis are often as valuable as the most expertly performed instrumental investigation

A method of eliciting a history, and examining a patient is as follows:

On admission to hospital:

- (a) Complete history.
- (b) Urinalysis and blood count
- (c) Kidney function tests (PSP and N.P.N.).
- (d) Roentgenograms and cystoscopy if indicated

Outline of History or Case Report

1 Chief complaint.

Secondary complaints in order of importance

2 History of present illness, onset, giving symptoms in chronological order as much as

possible, looking for points in history referable to the different systems in order, as in the physical examination.

Frequency:

- Day, night, on exercise.
- Effect of cold or dampness.
- Duration of symptoms.

Pain:

- Site of pain.
- Radiation to loin, genitals, bladder, perineum or penis.
- Character—sharp—dull—aching, throbbing, requiring morphia
- Duration.
- Number of attacks.

Hematuria:

- Color and character. bright red clots—old or new.
- Time of appearance—before, during, or after urination.
- Following exercise or intercourse
- Duration

Dysuria:

- Time: on exercise, before, during, or after urinating
- Radiation to penis or elsewhere.
- Duration.

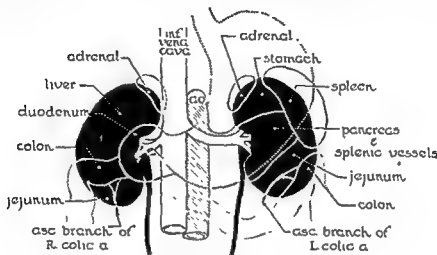
Alteration of Urinary Stream:

- Gradual or sudden narrowing of stream.
- Retention of urine, complete or incomplete.
- Number and duration of attacks
- Catheterization or other instrumentation employed.
- Hesitancy in onset; force.

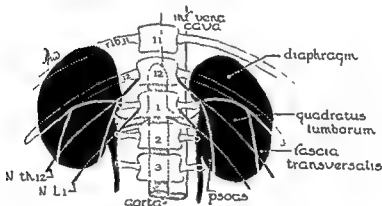
at the disc between the 11th and 12th thoracic vertebrae. The lower pole lies halfway between the transpyloric plane and the intertubercular line 3 inches from the midline; i.e., at the disc between the 3rd and 4th lumbar vertebrae. The left kidney is very slightly higher than the right.

POSTERIOR RELATIONS OF THE KIDNEYS

1. Diaphragm
2. 12th rib
3. Psoas muscle
4. Quadratus lumborum
5. Fascia transversalis
6. 12th thoracic nerve
7. 1st lumbar nerve
8. Tips of transverse processes of 1st, 2nd, and 3rd lumbar vertebrae.



Anterior Relations



Posterior Relations

Fig. 229—Anterior and posterior relations of kidneys.

The ureter is marked out by a line drawn from the hilus of the kidney to the pubic tubercle.

ANTERIOR RELATIONS OF THE KIDNEYS

| Right Side | Left Side |
|---|---|
| 1 Adrenal | 1. Adrenal |
| 2. Duodenum | 2. Stomach |
| 3 Liver | 3. Spleen |
| 4. Colon | 4. Pancreas |
| 5 Ascending branch of right colic artery. | 5 Colon |
| | 6 Ascending branch of left colic artery |

A capsule of fat surrounds each kidney and it in turn is enclosed between two fibrous layers derived from the fascia transversalis. The anterior layer is continuous with that of the opposite side and therefore passes in front of the inferior vena cava and the aorta. The posterior layer passes behind the kidney to the vertebral column. Above, the two layers unite. Below, the two layers are open.

- 5 Creatinine.
6. Red and white counts, hemoglobin.

Bacteriological examination of urine specimen:

- Sterile specimen for culture.
 Prepare stained smears including methylene blue; Gram's stain and Ziehl-Neelsen

Urethral discharge:

- Smears: methylene blue
 Gram's stain

Prostatic fluid:

- Microscopic
 Smear: methylene blue
 Gram's stain

Differential renal function tests:

- P.S.P.
 Urea Clearance.

Special Tests:

- Mosenthal, 3 glass specimen
 Urea clearance
 Guinea pig inoculation

KIDNEYS

Anatomy

Surface Anatomy.—The kidneys are marked out on the surface of the body by first drawing the vertebral column, marking in the various transverse or horizontal levels. These are the xiphi-

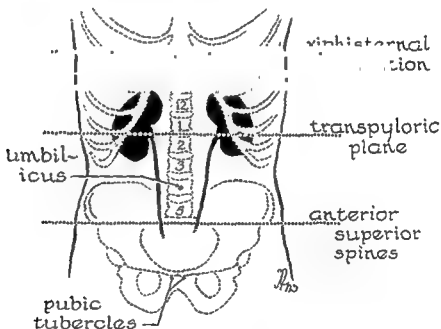


Fig 228 —Surface anatomy of kidneys.

Dark-field examination:

- Serum from sores, puncture of inguinal glands.

Cystoscopy:

- Ureteral urine specimens for:
 (a) Macroscopic, (b) Urea, (c) Volume,
 (d) Microscopic, (e) Culture, (f)
 Guinea pig inoculation.

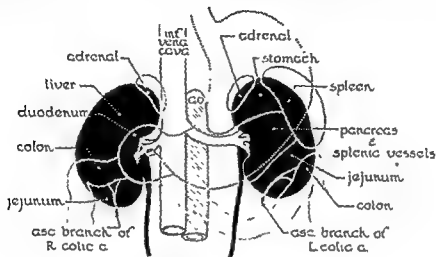
junction (between 9 and 10 T); transpyloric plane (between 1 and 2 L), umbilicus (middle of body of 4 L); anterior superior spines (between 5 L and 1 S).

The upper pole of the kidneys lies halfway between the horizontal line at the level of the body of the sternum and the transpyloric plane 2 inches from the midline,

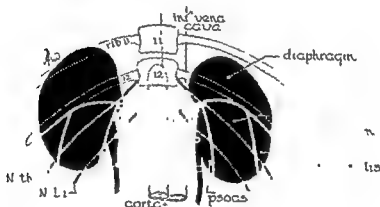
at the disc between the 11th and 12th thoracic vertebrae. The lower pole lies halfway between the transpyloric plane and the intertubercular line 3 inches from the midline; i.e., at the disc between the 3rd and 4th lumbar vertebrae. The left kidney is very slightly higher than the right.

POSTERIOR RELATIONS OF THE KIDNEYS

1. Diaphragm
2. 12th rib
3. Psoas muscle
4. Quadratus lumborum
5. Fascia transversalis
6. 12th thoracic nerve
7. 1st lumbar nerve
8. Tips of transverse processes of 1st, 2nd, and 3rd lumbar vertebrae.



Anterior Relations



Posterior Relations

Fig. 229—Anterior and posterior relations of kidneys.

The ureter is marked out by a line drawn from the hilus of the kidney to the pubic tubercle.

ANTERIOR RELATIONS OF THE KIDNEYS

Right Side

1. Adrenal
2. Duodenum
3. Liver
4. Colon
5. Ascending branch of right colic artery.

Left Side

1. Adrenal
2. Stomach
3. Spleen
4. Pancreas
5. Colon
6. Ascending branch of left colic artery

A capsule of fat surrounds each kidney and it in turn is enclosed between two fibrous layers derived from the fascia transversalis. The anterior layer is continuous with that of the opposite side and therefore passes in front of the inferior vena cava and the aorta. The posterior layer passes behind the kidney to the vertebral column. Above, the two layers unite. Below, the two layers are open.

In nephroptosis, the kidney descends between these two layers. A perinephric abscess likewise extends downward upon the psoas muscle between these two layers. Nephroptosis may be corrected by stripping the capsule off the upper half of the kidney

vidually into the inferior vena cava; on the left side, they all empty into the renal vein. The lymphatics from the right adrenal empty directly into the portal system. Those of the left side are connected with the lumbar glands and pass downward to the groin

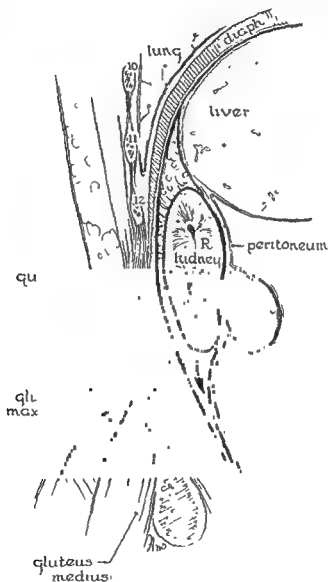


Fig. 230.—Perirenal fascia.

and attaching the lower pole to the 12th rib. It may also be corrected by suturing the anterior layer of the perirenal fascia to the posterior abdominal wall, thereby forming a basket under the kidney.

The three paired glands on the right side (adrenal, kidney, and testicle) empty indi-

vidually into the inferior vena cava; on the left side, they all empty into the renal vein. The lymphatics from the right adrenal empty directly into the portal system. Those of the left side are connected with the lumbar glands and pass downward to the groin

Embryological Development of Kidney.—In the human being there are three stages of development of the kidney. The pronephros, which develops during the second week of embryonic life on each side of the vertebral column, forms the Wolffian body, the caudal portion of which is tubular and extends distally to connect with the cloaca. About the third week of embryonic life degeneration of the pronephros and development of the mesonephric tubules occur simultaneously, the latter replacing the former.

which subdivide to form the minor calyces. Thus each major calyx and in turn each tubule has a cap of metanephric tissue which becomes more highly differentiated into excretory glomeruli and convoluted tubules.

Congenital Anomalies of the Kidneys

1. **Polycystic Kidneys.**—The renal glomeruli may, in part, fail to join the collecting tubules. If such closed glomeruli secrete urine, cysts are formed. Marked degrees of this condition give rise to congenital poly-

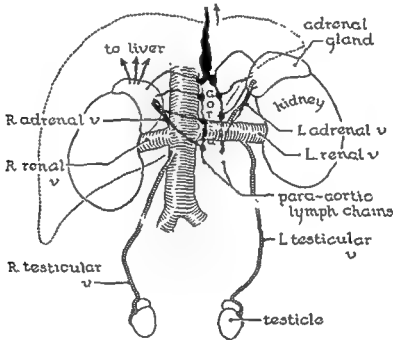


Fig 231 —Venous drainage of the three paired glands, and lymphatic drainage of the adrenals

At a later date the metanephros develops and becomes the permanent kidney. The excretory portion is developed from the caudal end of the nephrogenic cord (at the level of the first and second sacral segments).

The ureteric bud arises from the postero-medial wall of the mesonephric duct and eventually forms the entire tubular collecting system. The cranial end of the ureteric bud enlarges, comes into contact with the metanephros (excretory portion) which forms a cap around it, and then by a series of subdivisions forms the major calyces

cystic kidney. Minor degrees account for isolated or solitary cysts.

2. **Congenital Pelvic Kidney.**—During its development, the metanephros undergoes a relative change of position which is usually called the "ascent" of the kidney. This is partly the result of cephalad growth of the ureter and blastemal cap, but is due in part also to diminution of the body curvature during development. During ascent of the kidneys their intrinsic blood vessels receive their blood supply from lateral splanchnic stem arteries which arise from the aorta at increasingly higher levels until the final renal

artery is reached. Failure of ascent results in congenital pelvic kidney and abnormal vascular supply.

3 **Aberrant Renal Arteries.**—This is the result of the persistence of the blood supply from lower levels than normal.

Double ureter may be either unilateral or bilateral, complete or incomplete.

6 **Fetal Lobulation** may persist in adult life.

7. **Congenital Absence of One Kidney** is the result of the failure of the mesonephric

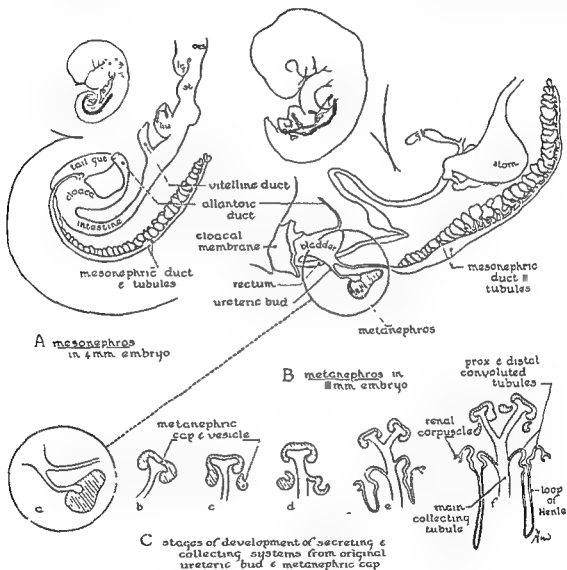


Fig. 232 —Mesonephric and metanephric tubules in the embryo showing development of collecting system (After Shukinami)

4. **Horseshoe Kidney** is due to fusion of the lower poles of the kidneys during their ascent.

5. **Double Ureter and Triple Ureter** are due to early division of the ureteric bud.

duct (Wolffian duct) to produce a corresponding renal bud.

8. **An Aplastic or Anaplastic Kidney** is due to failure of the metanephrogenic blastema to develop

TABLE X
EMBRYOLOGY AND CONGENITAL ABNORMALITIES

| Kidney | | | | | |
|--------------|--|------------------------------------|--|------------------------|---------------------|
| | | | sigmoid | unilateral fused | |
| Renal Pelvis | reduplication | hydronephrotic | bifid | extrarenal intrarenal | |
| Ureter | duplication complete incomplete | megaloureter stricture ureterocele | valves diverticulum twists & kinks | ectopic-onifice | |
| Blood Supply | aberrant vessels | (arterial and venous) | | low origin high origin | |
| Bladder | absence complete incomplete duplication | hypertrophy | diverticulum persistent cloaca | ectopic | urachal cyst |
| Urethra | absence complete incomplete duplication | hypertrophy | valves cysts diverticulum | | |
| Penis | absence complete incomplete duplication | hypertrophy rudimentary penis | hypospadias epispadias hermaphroditism | | |
| Testicle | anorchism monorchism polyorchism microorchism synorchism | infantile | | | cryptorchid ectopic |

Persistent anomalies of opposite sex are: Male—hydatid morgagni, utriculus masculinus. Female—Gartner's duct

TABLE XI
INCIDENCE OF 471 CONGENITAL ANOMALIES OF THE KIDNEY AND URETER IN 18,460
CONSECUTIVE UROLOGICAL ADMISSIONS TO ROYAL VICTORIA
HOSPITAL UP UNTIL 1940

| CONGENITAL ANOMALY | NO. OF CASES IN SERIES | PERCENTAGE IN SERIES | CLINICAL INCIDENCE | CLINICAL INCIDENCE REPORTED IN LITERATURE |
|--|---------------------------|-------------------------|-----------------------|---|
| Solitary kidney | 13 | 2.76 | 1:1420 | 1:1600 |
| Hypoplasia of kidney | 35 | 7.43 | 1:527 | 1:600 |
| Abnormal form of kidney | 58 | 12.31 | 1:318 | — |
| Polycystic kidney | 37 | 7.86 | 1:497 | 1:250 |
| Horseshoe kidney | 24 | 5.10 | 1:766 | 1:644 |
| Ectopic kidney | 19 | 4.03 | 1:972 | 1:1000 |
| Malrotation | 47 | 9.98 | 1:392 | — |
| Duplication of renal pelvis and ureter | 97 | 20.70 | 1:190 | 1:195 |
| Triple renal pelvis and ureter | | | — | 4 cases reported |
| Abnormality of pelvis and calyces | 79 | 16.67 | 1:234 | — |
| Abnormalities of the ureter | 15 | 3.18 | 1:1231 | 1:1500 |
| Valve in ureter | | — | — | 4 cases reported |
| Aberrant vessels | 47 | 9.98 | 1:392 | 1:313 |

9 Rotation of the Kidney—may be

- (i) absent (nonrotation);
- (ii) incomplete,
- (iii) excessive;
- (iv) reversed

Symptoms in the Congenital Anomalies Shown in Table XI.

| | Percentage | Approximate Fraction |
|------------------|------------|----------------------|
| Asymptomatic | 17% | 1/5 |
| Pain | 79% | 4/5 |
| Frequency | 52% | 1/2 |
| Hematuria | 25% | 1/4 |
| Calculi | 10% | 1/10 |
| Gastrointestinal | 32% | 1/3 |

The malformation of the kidney, as such, rarely causes trouble, unless it is complicated by secondary pathological changes. This finding has been supported by the large number of anomalous kidneys discovered at

autopsy in patients who were urologically asymptomatic during life.

Only 17 per cent of individuals who have a congenital anomaly reach adult life without developing symptoms referable to the malformation. It would seem to be the rule rather than the exception for the patient with an anomaly to develop symptoms that soon bring him to the urologist. Approximately 30 per cent will have symptoms referable to the gastrointestinal tract.

Practically any urological symptoms may be encountered in individuals with congenital anomalies. In most instances, the symptoms are due to acquired disease. There are many cases, however, in which there is no evidence of renal or ureteral disease, but the complaints are renal or ureteral in location.

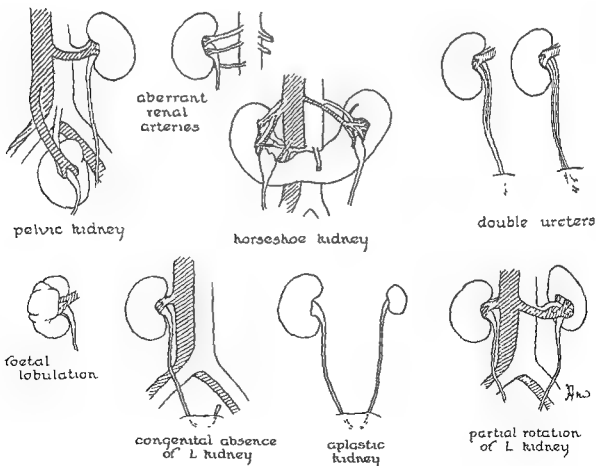


Fig. 233—Congenital abnormalities of the kidney and ureter (Modified after Grant)

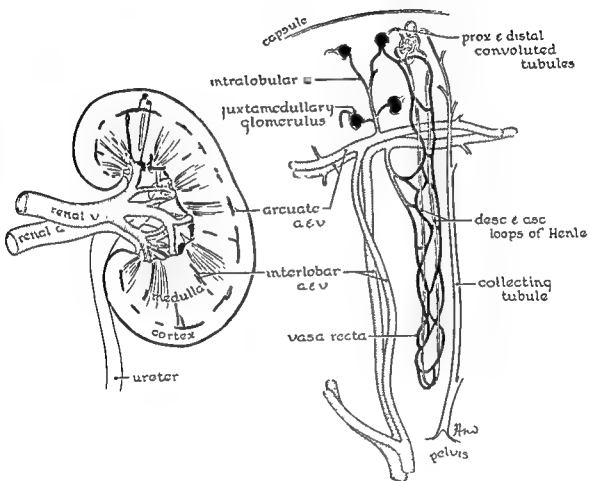


Fig 234.—Renal circulation.

The symptoms may be produced by

1. Irritation of adjacent nerves.
2. Increased peristalsis of a small, asymmetrical portion of the kidney pelvis.
3. Relative narrowing of the lumen at the point of bifurcation, and poorly coordinated ureteral peristalsis.
4. Physiological hypertrophy on the normal side, or in the solitary kidney itself.

Gastrointestinal symptoms may be produced by a distended renal pelvis sending impulses through the renal digestive reflex arc via the celiac plexus.

The associated pathological processes encountered in congenital anomalies are due to malposition, malrotation with ureteral obstruction, and resultant hydronephrosis. Infection occurs in 80% of cases. Almost every known type of secondary renal disease has been found in anomalous kidneys. The commonest associated pathological findings are pyelonephritis (60%), hydronephrosis (45%), and calculus (20%).

Thirty per cent of all the patients with a congenital anomaly of the urinary tract will require a major operative procedure, and of these 50% will require a nephrectomy.

Physiology of the Kidney

Renal Circulation.—Each kidney is supplied by a renal artery derived from the aorta. The artery enters the hilus of the kidney between the renal vein which is anterior and the renal pelvis which is posterior. Upon entering the kidney, or just prior to entry, the artery divides into several branches. These branches in turn give off interlobar arteries which run radially between the pyramids to the zone between the cortex and medulla, where they branch to form the arcuate arteries which run in a series of arcades between the two zones. From the arcuate arteries radial branches (intralobular arteries) are given off which supply the cortex while other branches extend downward into the medulla.

The veins accompany the arteries and form the same pattern. Each renal vein empties into the inferior vena cava.

It is now considered that there are three possible circulations within the kidney. The first is that just described and represents the normal circulation. The second, or inner, circulation, as described by Trueta and his associates, consists of the passage of blood through the interlobular artery, the glomeruli lying in the deepest zone of the cortex (juxtamedullary glomeruli), arterial components of the vasa recta in the medulla, the venous components of the vasa recta, and the interlobular vein. Trueta and his associates claimed that during conditions of stress, blood might be "shunted" through this inner circulation resulting in blanching and relative ischemia of the cortex. There is no proof to date that this occurs clinically.

The third circulation within the kidney has been postulated by Barrie. He has described smooth muscle bundles which run longitudinally and parallel with the arcuate arteries, but on only one side of these arteries. They branch when the arteries branch, but are not continued onto the cortical arteries. Barrie has found a mesh of sinusoids or vacuoles in these muscle fibers. The sinusoids at one end have open communications with the arteriolar coil, and at the other end drain into one of the larger veins. The function of this system is unknown, but Barrie believes that it may be regarded as an arteriovenous shunt, which acts as a decompression mechanism when the arterial circuit is overloaded.

Glomerular and Tubular Function.—Renal function can be adjusted to meet the varying chemical demands placed upon it. By this means the composition of body fluids is maintained at levels consistent with normal cellular activity. Patients with impairment of renal function can maintain well-being only as long as they can be pro-

ected from stresses that tend to seriously alter the body chemistry. The normal volume of renal blood flow is approximately 1,000 c.c. per minute ($\frac{1}{5}$ of the cardiac output), so that in 5 minutes the total blood volume has passed through the renal circulation.

Filtration pressure is normally 25 mm. of mercury. It is the net effective pressure of two opposing forces. On the one side of the scale is a positive pressure of 75 mm. of mercury within the capillaries which tends to force water out of the capillaries. Opposing this there are pressures that tend to prevent filtration, as shown in Table XII.

process may be reversed, but if prolonged, it becomes irreversible.

Tubular Secretion and Reabsorption.—As indicated in Fig. 235, water, glucose, sodium chloride, carbon dioxide, urea, and potassium are reabsorbed by the tubule from the glomerular filtrate. Ammonia, hydrogen, and hippuric acid are added to the glomerular filtrate by secretion from the tubule. Urea, uric acid, and sulphates are not absorbed.

Table XIII shows the changes in concentration of the various constituents in plasma and urine, and the degree to which they are concentrated.

TABLE XII

| POSITIVE PRESSURE FAVORING FILTRATION | PRESSURES OPPOSING FILTRATION | NET EFFECTIVE FILTRATION PRESSURE | FILTRATE |
|---|---|---|---|
| 75 mm Hg derived from arterial side | (i) Osmotic pressure of plasma proteins 30 mm Hg. (ii) Interstitial pressure 10 mm Hg (iii) Pressure necessary to move fluid down tubule—10 mm Hg | 25 mm Hg | 120 c.c. of fluid per minute in tubule all except 1 c.c. of which is reabsorbed by the tubule |

TABLE XIII

CHANGES IN CONCENTRATION OF VARIOUS CONSTITUENTS IN PLASMA AND URINE

| | BLOOD PLASMA % | URINE % | CHANGE IN CONCENTRATION |
|-----------------------------|----------------------|------------|----------------------------|
| WATER | 90-93 | 95 | } not excreted |
| PROTEINS, FATS AND COLLOIDS | 7-9 | none | |
| GLUCOSE | 0.1 | traces | |
| Na | 0.30 | 0.35 | } concentrated slightly |
| Cl | 0.37 | 0.60 | |
| Ca | 0.008 | 0.15 | |
| Mg | 0.0025 | 0.006 | |
| Uric Acid | 0.004 | 0.05 | } moderately concentrated |
| K | 0.02 | 0.15 | |
| PO ₄ | 0.009 | 0.006 | |
| NH ₃ | 0.001 | 0.004 | |
| SO ₄ | 0.002 | 0.18 | } concentrated slightly |
| Urea | 0.03 | 2.0 | |
| Creatinine | 0.001 | 0.075 | |

Glomerular filtration ceases and anuria develops when the systolic blood pressure drops to below 60 mm. mercury. If hypotension persists for only a short while, the

Nerve Supply and Physiology of Kidney Pelvis and Ureter

The kidney capsule is supplied with sympathetic nerve fibers from T12 and L1.

Likewise the pelvis and ureter have a sympathetic nerve supply from the T11 and 12 and 1st lumbar segments. In the latter case the autonomic ganglia are situated in the adventitia of the ureter. So far as is known, the parenchyma of the kidney has no nerve supply. Overdistention of the renal pelvis, or back pressure and distention of the whole kidney, with consequent stretching of the capsule, causes pain. However, the parenchyma may be completely destroyed as in tuberculous caseation, without pain, since the parenchyma lacks a nerve supply. Parasympathetic fibers from the vagus enter the kidney, but their function is unknown.

lus for ureteral peristalsis is the production of urine by the kidneys and consequent distention of the ureteral wall. Peristalsis is increased by increased excretion of urine, by increased intra-abdominal pressure, and by ureteral obstruction.

Lymphatics of the Kidneys.—There are two sets of lymphatics associated with the kidneys. The superficial group of lymph channels lies in the superficial fat surrounding the kidney. The second or deep group of lymphatics lies within the substance of the kidney, surrounding the tubules and blood vessels in the interstitial tissue. They drain into larger channels at the hilus of the kid-

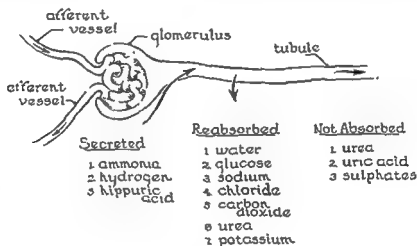


Fig. 235—Tubular secretion and reabsorption

The renal plexus which surrounds the renal artery consists of an intricate network of nerve fibers derived from the aorticorenal ganglia, the middle and inferior splanchnic nerves, intermesenteric nerves and fibers from the lumbar sympathetic chain. Surgical stripping of the renal artery, as sometimes carried out in selected cases of hypertension, is usually not a complete denervation.

The origin of the impulse which initiates ureteral contraction is obscure. Some workers believe that the impulse originates in the central nervous system. Others believe that the ureters can function automatically and initiate their own muscular contractions. Most agree, however, that the usual stimu-

ney and empty into adjacent aortic lymph nodes. The two systems intercommunicate freely, thus assisting the spread of infection through the kidney.

In retrograde pyelography, injection of excessive amounts of contrast media into the renal pelvis may cause a backflow of the dye into the deep (intrarenal) lymphatic system or veins. This is known as *pyelovenous backflow*.

Chyluria.—Normally the fat particles which enter the intestinal lymphatics give the lymph fluid a milky appearance (chyle). This passes upward through lymphatic channels and eventually into the main lymphatic duct. In some conditions such

as filariasis, the main lymphatic channels may become occluded. The chyle backs up and may be forced by ever mounting pressure into the deep lymphatics of the kidney and thence by further pressure into the renal pelvis, where it is excreted in the urine. This is known as *chyluria* and is the reverse process of pyelovenous backflow.

Orthostatic Albuminuria

Orthostatic albuminuria is a condition in which albumin is found in the urine without any pathological condition in the kidneys. It is usually due to lordosis causing some pressure on the renal vein resulting in delayed emptying.

Orthostatic albuminuria may be tested for by producing exaggerated lordosis in one of two ways—(1) By placing a pillow under the small of the back when the patient is lying flat; or (2) by having the patient bend backward 10° for 10 minutes while standing with his hands on hips.

The following criteria are required before a diagnosis of orthostatic albuminuria is made:

- 1 No past history of renal or cardiovascular disease

- 2 No elevation of blood pressure.

- 3 No W.B.C., R.B.C., or casts in the urine, except intermittently and in small numbers

- 4 Normal kidney function (P.S.P., urea clearance test, urine concentration tests).

- 5 Normal blood chemistry (N.P.N., urea clearance, total protein, normal albumin-globulin ratio)

- 6 Negative plain x-rays and normal intravenous pyelograms.

- 7 Absence of albumin in the urine secreted when in the horizontal position.

Hydronephrosis

Hydronephrosis is a dilatation of the renal pelvis with stagnation of urine caused by an obstruction to the outflow of urine. The obstruction is nearly always mechanical, but on rare occasions may be due to neurogenic

dysfunction. Hydronephrosis may affect one or both kidneys; it may be congenital or acquired, and may be infected or not infected.

Primary Renal Atrophy.—Sudden complete obstruction of the ureter is thought by some to produce primary atrophy of the kidney without a preceding hydronephrosis. This belief is, however, not borne out by either experimental or clinical observation. It occurs so rarely that it may be disregarded.

Hydronephrosis With Secondary Renal Atrophy.—The usual sequence of events following obstruction to the outflow of urine is as follows. Dilatation of the renal pelvis, or renal pelvis and ureter above the site of the obstruction occurs, whether this is at the ureteropelvic junction, in the ureter, or at the entrance of the ureter into the bladder. As the back pressure is increased, the walls of the ureter and pelvis become progressively distended. Urine continues to be formed. Absorption of urine from the pelvis occurs while the kidney still continues to secrete urine. The contents of the hydronephrotic sac thus change continuously. Absorption of the fluid in the hydronephrosis is thought to take place either through the tubules or by pyelovenous backflow, or by both.

Eventually the combination of increased intrapelvic pressure, renal ischemia and disuse, results in degeneration and atrophy of the kidney.

Hydronephrosis develops more rapidly in a high obstruction than it does in a low obstruction. The rate of urine formation, however, remains the same whether fluids are forced or withheld completely.

Symptoms.—The symptoms are those of the underlying pathological process, i.e., obstruction. There may be no symptoms at all or a dull ache in the back. If the hydronephrosis becomes infected, there will be chills, fever, and frequently pus in the urine.

Treatment.—Remove the underlying cause of the hydronephrosis if possible. If this is not possible, or if the disease in the kidney is far advanced, remove the kidney.

Anuria

Anuria is the cessation of urine formation. Oliguria is decreased secretion of urine. The causes of anuria are listed below. The post-renal causes are due to obstruction to the outflow of urine. They are usually classified under anuria because no urine is obtained. In these cases urine is formed so that it is not a true anuria

CAUSES OF ANURIA

I. Pre-Renal (Circulatory).—

1. Occlusion of the main vessels of both kidneys by embolism or by the external pressure of a tumor.
2. Reduction of blood volume due to—
 - (i) Dehydration, e.g., diarrhea, vomiting, excessive perspiration.
 - (ii) Hemorrhage.
3. Retention of fluids in the tissues (generalized edema).
 - (i) Advanced cardiac decompensation.
 - (ii) Retention of salt in the tissues
4. Endocrine form—due to dysfunction of the pituitary, thyroid or adrenal gland. These forms are rare
5. Low blood pressure, as in shock.
6. Reflex anuria—or inhibitory form
 - (i) Inhibition of uninvolved kidney by obstruction in opposite kidney.
 - (ii) Vascular spasm of main vessels following splanchnic stimulation as in abdominal surgery, distention, etc.
 - (iii) Peripheral irritation, e.g., passing sounds, catheterization, ureteral calculus, etc.
 - (iv) Anesthesia—especially spinal.
 - (v) Severe painful stimuli.

II. Renal—Intrinsic Renal Disease—(Excretory).—These occur on the basis of inadequate secreting renal tissue.

1. Acute infectious nephritis, glomerulonephritis, suppurative nephritis.
2. Extensive destruction of kidney tissue bilaterally, e.g., advanced tuberculosis, polycystic kidney disease.
3. Acute nephrosis, e.g., poisoning by heavy metals, mercury, bismuth, arsenic, lead.
4. Acute toxic nephritis.
 - (i) Toxic products from damaged liver.
 - (ii) Pre-eclampsic toxemia.
5. Postpartum cortical necrosis.
6. Following removal of solitary kidney.

III. Post-Renal—Obstructed Outflow of Urine (Eliminatory)

1. *Intrinsic*—blockage of one or both ureters by blood clot, calculi, stricture, neoplasm of pelvis or ureter, sulfonamides, tumor of bladder, enlargement of prostate.
2. *Extrinsic*.
 - (i) Accidental injury or ligation of ureters during gynecological or other operations.
 - (ii) Retroperitoneal growths and malignant pelvic tumors.

IV. Composite or Mixed

This group includes those cases where more than one of the above factors are present.

1. Injuries to kidney and ureter.
 - (i) Injury or avulsion of a single kidney.
 - (ii) Bilateral renal rupture.
2. Lower nephron nephrosis—which may occur after crushing injury to muscle, nontraumatic muscular ischemia, burns, transfusion reactions with incompatible blood, heat stroke, malaria, toxemia of pregnancy, alka-

losis, sulfonamide anuria, and following transurethral prostatic resection.

TREATMENT OF ANURIA

I. Treatment of the prerenal and renal causes of anuria consists of:

1. *Prevention*—maintain an adequate blood volume and adequate blood pressure.

2. *Active treatment*—try to restore the blood pressure and blood volume to normal by means of blood transfusions, vasoconstrictors, adrenal cortical extracts. Renal decapsulation may be indicated.

II Treatment of postrenal causes of anuria: Remove obstructive cause if possible.

III. Treatment of lower nephron nephrosis: In lower nephron nephrosis the kidney normally tends to recover spontaneously between the 5th and 9th day of the disease. During this period it is important to limit fluids to 1,500 c.c. daily, i.e., the amount lost through respiration and perspiration. If the anuria persists and the kidney fails to recover spontaneously, the artificial kidney may be used to wash the waste products from the blood.

Sulfonamide Anuria

The use of the sulfonamides either as a prophylactic or therapeutic measure entails some risk.

The sulfonamides are excreted by the kidneys, being concentrated as much as three hundreds times in the urine by tubular absorption. There is no relationship between the blood level of the sulfonamide and the patient's ability to tolerate the drug.

Any patient receiving the sulfonamides should be given enough fluids to insure a twenty-four hour urinary output of at least 1,500 m.c. Sodium bicarbonate may be given along with the sulfonamide.

Sulfonamide anuria can be prevented by limiting the administration of sulfonamides and controlling the pH of the urine. The

blood and urine levels of the drug, the presence of microscopic hematuria, and the possibility of sensitivity to the drug should all be borne in mind when using this type of therapy.

When anuria does occur, treatment should follow a definite plan. The drug must be discontinued. Cystoscopy is done to exclude the existence of mechanical obstruction to the drainage of urine and to wash each kidney pelvis, thereby removing any crystals and blood clot present. If mechanical obstruction of the ureter exists, and it is impossible to catheterize either renal pelvis, pyelotomy must be performed.

Renal Hypertension

See Chapter on Surgical Treatment of Hypertension

Renal Rickets

Renal rickets is a disease of childhood characterized by the symptoms of polyuria, polydipsia, retardation of growth, deformity of growth, and delayed sexual development. A genu valgum develops. With the increased softening of the metaphyses, extreme degrees of distortion and deformity may be produced.

The kidneys show some degree of deficiency which may be pronounced as the disease advances. Chronic pyelonephritis is frequently present and calculus formation is a frequent complication. The specific gravity of the urine is fixed at a low level.

There are two types of bone changes which may occur—

1. Rachitic changes at the ends of long bones.

2. Generalized osteoporosis with loss of distinction between cortex and medulla.

The blood phosphate is elevated and the blood calcium lowered.

The cause of renal rickets is unknown. One theory is that it is primarily a renal disease, due to inability of the kidney to excrete an adequate amount of phosphates. A second theory is that renal rickets is an

endocrine disease, caused by primary over-activity of the parathyroid glands with or without associated pituitary dysfunction.

There is no known treatment for this disease. Death from renal failure usually occurs before the age of 20 years.

RENAL INJURIES

The kidney is protected by its mobility and retroperitoneal position. Injuries are caused by external violence, such as a fall, kick, or blow on the back or side; by crush-

2. Contusion of the kidney. This is not serious unless it becomes infected.

3. Rupture of the parenchyma, usually posterior surface, with resultant pulping of the kidney. Hemorrhage may be marked or even fatal. Hematuria is always present. Nephrectomy may be considered.

4. Rupture of the parenchyma and capsule. The hemorrhage is greater than when the capsule is not torn.

5. Rupture of the parenchyma, capsule, and pelvis. The vessels of the renal pedicle

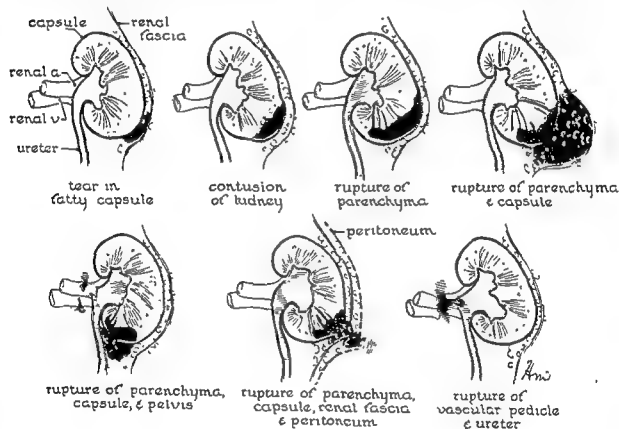


Fig 236—Injuries of the kidney.

ing injuries or penetrating wounds. Eighty per cent are due to blows on the back or side. The kidney responds like a ball of fluid to the force of an impact, transmitted in all directions.

The pathological changes that occur are shown in Fig. 236 and are tabulated as:

1. Tears of the fatty capsule only. Hemorrhage into the perirenal tissues may be absorbed, organized, or encysted.

are usually torn, and hemorrhage may be fatal. There may be urinary extravasation with infection.

6. Rupture of the parenchyma with rupture of the peritoneum. This is a rare but serious type which occurs chiefly in children.

7. Rupture of the vascular pedicle and tearing of the ureter. The pedicle is sheared off. Death occurs promptly from hemorrhage.

8 Rupture of the liver or spleen may be associated with rupture of the kidney.

Diagnosis is based on (1) history of injury, (2) variable degree of shock, (3) hematuria, (4) localized tenderness and rigidity, (5) a palpable mass may or may not be present

Treatment.—1 *Immediate.* The following supportive measures are most important: (a) treatment of shock, (b) adequate blood transfusion. The blood pressure, pulse, and degree of hematuria must be carefully followed.

2 *If patient is recovering* (a) continue conservative treatment, (b) give urinary antiseptic daily; (c) do an intravenous or retrograde pyelogram when the patient is out of shock, to obtain an approximate idea of the degree of damage to the kidney, and to establish the presence of the other kidney

3 *If condition continues to deteriorate after initial therapy, i.e., pulse rate steadily increases or blood pressure cannot be maintained in spite of continuous transfusion,* (a) prepare for nephrectomy and carry out exploration of the kidney, (b) make certain the opposite kidney is present before operating. This may be done by intravenous pyelography (c) When the kidney is exposed, if attempts to control the bleeding are unsuccessful, *immediate nephrectomy* should be done

Complications.—

1 Sepsis—usually prevented by giving urinary antiseptics or antibiotics

2. Secondary hemorrhage

3 Fibrosis of part or the whole kidney resulting in scar formation with partial or complete loss of function

It is for this reason that it is advisable to do periodic differential phenolsulfonphthalein tests during the period of recovery.

Prognosis.—

1. Seventy per cent recover without requiring an operation, even though critically ill on admission

2. A secondary nephrectomy for a non-functioning kidney may be required months after the initial injury.

NEPHROPTOSIS

Nephroptosis of the kidney is descent of the kidney beyond the normal 4 cm. diaphragmatic excursion. Reference to Fig. 230 will indicate how this may occur. It is present in approximately 1% of men and 6% of women. It is much more common on the right side because of the weight of the liver and the fact that the renal fossa is shallower on this side. The kidney is normally held in place by the fatty capsule and surrounding fascia, the vascular pedicle, and the support supplied by surrounding organs. In a thin person, and in one in whom there is a decrease of muscle tone, the additional trauma of a twist or fall, or the added weight of an overloaded colon, may be sufficient to cause ptosis of the kidney. Ptosis frequently causes kinking of the ureter and back pressure with resultant hydronephrosis

The symptomatology varies considerably. There may be no symptoms referable to the ptosis, but in 66 per cent of cases there is pain in the lumbar region, due to improper drainage which gradually increases as the day wears on, and is relieved by recumbency, which permits the kidney to fall back into place and thus improves drainage. The symptoms may be entirely gastric (22%), because of a reflex from the kidney through the celiac axis. The patient then complains of epigastric pain, nausea, and vomiting. The symptoms are occasionally entirely nervous in type, the patient being irritable and easily fatigued. It is of the utmost importance to select cases for operation carefully. It is only those patients who have pain in the back, increasing as the day advances, and relieved by lying down who should be subjected to operation. The accidental finding of ptosis of the kidney without any of the above symptoms is definitely not an indication for surgery. Similarly,

the emotionally unstable type of individual with innumerable complaints, will not obtain a good result from nephropexy, even though the corrected anatomical position of the kidney is excellent; he will continue to complain of pain. Generally speaking it is advisable to treat these patients with a well-fitting elastic girdle with attached pad.

In the patient selected for operation, nephropexy may be done by one of several procedures. Decapsulation of the upper two-thirds of the kidney with a suture passed through the lower pole and tied over the 12th rib is the operation most used. The

currence of the ptosis does not take place. If, however, all cases of nephroptosis are operated upon without selection, the results will not be uniformly good.

TUMORS OF KIDNEY

A. Tumors of Children

1. Wilms' Tumor (synonym: embryoma or adenomyosarcoma).

Clinically, Wilms' tumor or embryoma represents 20% of all tumors occurring among children. The age incidence is from birth to 10 years. Seventy-five per cent oc-

TABLE XIV
CLASSIFICATION OF TUMORS OF THE KIDNEY

| A CHILDREN—Wilms' embryoma | | | |
|------------------------------------|--|--|---|
| B ADULTS | | | |
| (a) Parenchyma Tissue of Origin | | | |
| 1. Epithelial | Benign: Adenoma Malignant: Hypernephroma Carcinoma | | Synonym Grawitz tumor Hypernephroid carcinoma Adenocarcinoma |
| 2. Connective Tissue | Benign: { Fibroma Lipoma Leiomyoma Angioma Malignant: Sarcoma | | |
| 3. Wilms' embryoma | Embryoma Adenomyosarcoma | | |
| (b) Renal Pelvis | Papillary { Papilloma Papillary carcinoma Solid type { Squamous cell carcinoma Adenocarcinoma | | |
| (c) Capsular tumors | { Lipoma Fibrolipoma Fibrosarcoma Liposarcoma | | |

denuded area of the kidney becomes firmly adherent to the undersurface of the diaphragm, peritoneum, and posterior abdominal wall. An alternative procedure is to expose the kidney and use the perirenal fascia to form an apron, which is stitched down to the muscles of the posterior abdominal wall with interrupted sutures, thus forming a basket beneath the lower pole of the kidney. If cases are properly selected as indicated above, and if the operation is well performed, the results are excellent. The patient obtains complete relief of pain, and re-

cur in the first 5 years of life. In the later decades where other tumors are more common, one rarely encounters a Wilms' embryoma. Sex is not a factor. These tumors are extremely malignant. With early recognition, a 5-year cure may be effected in 25% of cases. A few cases have been reported in which the patient was alive 10 years or more after nephrectomy.

Origin.—The origin of mixed renal tumors is not clear. Most observers agree that the tumors are embryonal, in that they arise in the region of the developing kidney.

Three Theories of Origin.—

I. The tumor is due to inclusions of Wolffian tissue which have become displaced and which persist among the cells of the developing kidney or metanephros.

II The tumors arise from aberrant cells of the myotome and sclerotome, the apparent mixed character being explained by the varying constituents which enter the ultimate formation.

III The tumors are not due to inclusions from extrarenal sources, but they are true kidney, the embryonic tissue persisting and becoming metamorphosed into cellular structures of various types.

presence of muscle elements indicates that tissue from the myotomes is included. The small papillary buds seen in some of the glandular components of the tumor may be abortive glomerular structures

Signs—Symptoms.—

1. Presence of swelling in the upper abdomen—most cases.

2. Painless—usually.

3. Occasionally, painful—usually late—due to pressure or to tension on peritoneum associated with weakness, pallor, fever, gastrointestinal symptoms due to bowel displacement, hematuria (10%) and hypertension.

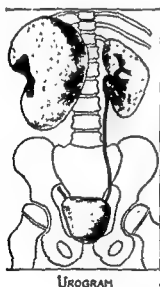


Fig 237—Wilms' embryoma, pyelogram and gross specimen

Pathology.—Gross: The Wilms' tumor is a large, solid, greyish white, encapsulated tumor of variegated histology. It is usually separated from the kidney by a layer of tough connective tissue. The capsule of the tumor blends with that of the kidney and with this membrane

Microscopic: The tumors are composed of a variety of tissues. The microscopic picture varies according to the predominating type of tissue present. In addition to simple epithelial and mesenchymal components, the

Diagnosis.—History, signs and symptoms as above, plus deformity of the pyelograms

Differential Diagnosis.—

1. Other kidney tumors and hydronephrosis.
2. Polycystic kidney disease.
3. Retroperitoneal tumors separate from the kidney.
4. Adrenal neuroblastoma (preoperative differential diagnosis may be impossible).

5. Splenic enlargements.
6. Tumors of liver.
7. Omental and pancreatic cysts
8. Ovarian tumors.

Treatment.—Preoperative radiation followed by nephrectomy offers the best results. The tumor usually shrinks with radiation. Nephrectomy followed by radiation is an alternative method of treatment.

Prognosis.—Although the tumor can be removed successfully, the likelihood of recurrence is so great that the prognosis is poor. Metastases may occur even before one suspects the presence of a primary tumor.

ally at postmortem in arteriosclerotic kidneys. They occur in the form of multiple discrete, whitish nodules located just beneath the renal capsule, or scattered throughout the renal parenchyma. They often occur bilaterally. Histologically, they are made up of irregular groups of epithelial cells ranged around lumina which are regular in outline. Papillary projections of epithelial cell masses into such a lumen are a frequent observation. The epithelium, however, is present only in a single layer. The resemblance to normal renal tubules is so close as to render differentiation difficult.

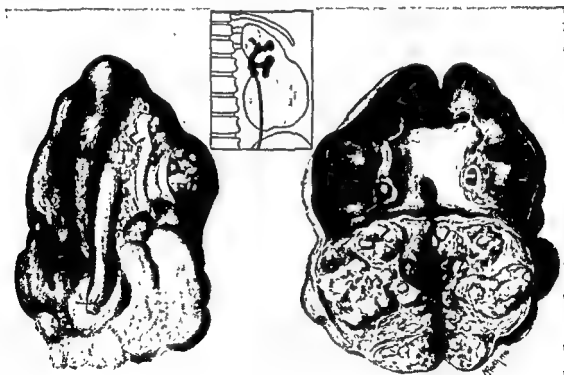


Fig. 238—Renal carcinoma showing extension into renal vein

B. Tumors of Adults

- (a) Tumors of Renal Parenchyma
 - (b) Tumors of Renal Pelvis
- Form 0.5% of all tumors in adults.

Tumors of Renal Parenchyma.—

I. TUMORS OF EPITHELIAL ORIGIN:

- (a) *Benign*: Adenoma.

Small adenomas, varying in size from 2 mm. to 1 cm. or larger, are found occasion-

- (b) *Malignant*—Carcinoma (Synonyms: Grawitz tumor; hypernephroma; hypernephroid carcinoma).

Primary malignant tumor of the kidney is almost always unilateral.

In 1883 Grawitz proposed the theory that these tumors arise from aberrant adrenal tissue. Most people today believe that the tumor is a renal carcinoma arising either from adult tubules, or from islets of nephro-

Three Theories of Origin.—

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II The tumors arise from aberrant cells of the myotome and sclerotome, the apparent mixed character being explained by the varying constituents which enter the ultimate formation.

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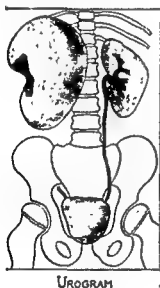


Fig 237—Wilms' embryoma, pyelogram and gross specimen.

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3. Retroperitoneal tumors separate from the kidney.
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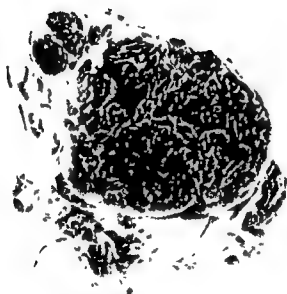


Plate XXXI.—Renal Carcinoma in Upper Pole of Kidney.

(Courtesy Victor F. Marshall. The Diagnosis of Genito-Urinary Neoplasms, American Cancer Society, Inc.)

genic tissue which have persisted in the renal cortex. A single renal tumor often exhibits mixed features.

Gross Pathology of Renal Carcinoma (Hypernephroma).—The striking variegated yellow and red of its gross appearance result from the high lipid content of the neoplastic cells and the hemorrhages which are almost invariably present.

Microscopic Pathology.—The clear cells which are prominent in microscopic sections of these tumors bear a vague resemblance to

growths may metastasize widely through the renal vein. It is therefore important to examine the vessels grossly and microscopically.

Metastases.—Metastases occur by direct extension of the growth through the capsule or into the renal vein, with showers of emboli into the blood stream. Spread takes place in this manner to the lungs, brain, and long bones. Pathological fracture of a bone may occur before the primary tumor is recognized.



Fig. 239—X-ray of chest showing cannon ball metastases from carcinoma of kidney

the cells of the adrenal cortex, which led Grawitz to believe that they were derived from adrenal rests. Many hypernephroid tumors are not composed entirely of clear cells. Granular cells and mixed cell variants are also recognized. Furthermore, not all hypernephroid tumors are malignant, and the differentiation between adenoma and adenocarcinoma is often difficult. The size of the tumor is not a criterion of its degree of malignancy. If there is obvious invasion of the pelvis or frank cellular anaplasia, then the diagnosis of malignancy is simple. However, encapsulated and benign-looking

Prognosis.—The prognosis is poor. Without surgical intervention a fatal outcome is inevitable within approximately one year. In cases seen before metastases are present, surgical removal offers a distinct hope of cure. The outcome depends upon an early diagnosis and complete removal of the tumor. In the majority of cases the disease is well advanced before the patient consults a doctor.

II. CONNECTIVE TISSUE TUMORS:

(a) Benign:

1 *Fibroma.* Two forms occur: The first as small whitish nodules which rarely cause

symptoms and are usually discovered at autopsy. The second form consists of large cortical growths which produce symptoms and require surgical treatment. They are rare, but may occur at any age, even in children.

2. *Lipoma*. Small multiple masses of lipid tissue may be present beneath the renal capsule or in the cortical substance. They are rarely larger than 1 cm. in diameter.

3. *Leiomyomas*. Leiomyoma is a tumor composed of plain muscle. It may have fibrous tissue mixed with it so that the tumor is in reality a fibroid, and identical to a fibroid in the uterus. It rarely reaches sufficient size to produce symptoms.

Sarcomatous degeneration may occur in any of the above tumors, but is rare.

4. *Hemangiomas* are occasionally encountered. Unless they attain considerable size and cause pressure or other symptoms, they are of no clinical importance. Hematuria does not occur unless the tumor erodes the renal pelvis. In the differential diagnosis of essential hematuria or occult hematuria, a minute angioma which may be so small that it is easily overlooked at operation, is a possibility to be considered.

(b) *Malignant:*

Sarcoma is very infrequent in adults. Fifty per cent occur between the ages of 40 and 60 years. The histopathological structure varies considerably, but all are highly malignant and invariably terminate fatally regardless of treatment. Spindle cell sarcoma is the most common type, with fibrosarcoma, leiomyosarcoma and other forms being very rare.

The symptomatology, diagnosis, and treatment are the same as for other parenchymal tumors.

Signs and Symptoms of Renal Tumors.—

1. *Hematuria* is the most constant finding and may be the earliest symptom except in Wilms' tumor in children in whom a mass is usually the first sign. Bleeding is always intermittent and seldom profuse. A single

hemorrhage may be followed by months or even years without recurrence.

2. *Pain* in the kidney is the initial symptom in 20% of cases and is present in a large percentage of the late cases. It may be caused by stretching of the renal capsule by the expanding tumor, or it may result from pressure by the mass on neighboring nerves, viscera, or ureter, i.e., hydronephrosis.

3. Although hematuria and pain are the commonest symptoms, loss of weight, weakness, gastrointestinal disturbances, fever, cachexia and anemia may occur. These are usually late symptoms.

Diagnosis.—

The diagnosis of renal tumor is made on (1) history, signs and symptoms, (2) deformity shown in the pyelogram, usually a filling defect.

Prognosis.—Success in the treatment of renal tumors depends upon their early recognition. The over-all prognosis is poor.

Treatment.—1. Prompt nephrectomy with postoperative radiation is the treatment of choice.

2. In inoperable cases with considerable bleeding, Dicumarol may be given to diminish coagulation and minimize the colic caused by the passage of blood clots.

3. Ligation of the ureter may be carried out if the tumor is inoperable and the kidney is not infected.

Tumors of Renal Pelvis.—Five per cent to 7% of all tumors of the kidney are primary in the renal pelvis.

| | |
|------------------------------------|---------------------------|
| 75% are Papillary | { Papilloma—benign |
| | { Papillary carcinoma |
| 25% are Nonpapillary or solid type | { Squamous cell carcinoma |
| | { Adenocarcinoma |

Connective tissue tumors are exceedingly rare.

Carcinomas of the renal pelvis arise from the epithelium which is similar morphologically to that of the ureter and urinary bladder. The epithelium of the renal pelvis is stratified and does not exhibit keratinization or cornification. It is therefore called transitional in type. Certain stimuli, such as chronic infection and stones, may cause the

tom. Pain along the course of the ureter may occur. A mass along the course of the ureter may be palpable.

Diagnosis.—The diagnosis is made on the history of physical signs and deformity shown in the pyelograms or ureterograms.

Treatment consists of nephroureterectomy where possible. If this is not possible, intra-ureteral radium or deep roentgen therapy may be used.

Prognosis.—The prognosis in ureteral tumors is good if they are diagnosed early.

PYOGENIC INFECTIONS OF THE URINARY TRACT

Pyogenic infections of the urinary tract are due to invasion of the kidney, ureter or bladder by the organism itself, rather than by a toxin. The organism causes an inflammatory reaction in the interstitial tissue whereas the toxin acts on the glomerulus producing a glomerulonephritis.

Diagnosis.—Certain criteria must be met to establish an accurate diagnosis. Pyogenic organisms must be present. Failure to examine the specimen for organisms is the commonest cause of error in diagnosis. The specimen examined should be freshly voided in the male and be a catheterized specimen in the female. It should be collected in a sterile container. If organisms are found, they may then be assumed to come from the patient. The prognosis depends largely upon the etiology.

Bacteriology.—From a clinical point of view an elaborate process of staining is undesirable. A Gram stain is sufficient to identify a bacillary infection from a staphylococcal, streptococcal, or mixed infection. In 90% of cases this is all one needs to know. If the infection is one of the rarer types which does not respond to treatment, a culture can then be done to identify the organism. The process of examining stained smears of urinary sediment can be further simplified by doing a methylene blue stain instead of a Gram stain, since only 2 per cent of the infections are caused by gram-positive organisms.

The infecting organisms in the order of frequency are:

- 1 *B. coli* (*Escherichia coli*) 42%
- 2 Other gram-negative bacilli 30%

Roughly 70% are due to gram-negative bacilli.

- i. *Aerobacter aerogenes*. (*B. coli aerogenes*).
- ii. *Pseudomonas aerogenes*. (*B. pyocyaneus*).
- iii. *Pseudomonas fluorescens*. (*B. fluorescens*).
- iv. *Proteus vulgaris*. (*B. proteus*).
- 3 i. *Staphylococcus pyogenes* 12% } 20%
- ii Micrococci 8% }
4. Mixed organisms
- 5 Other types
 - i. Gram-positive bacilli
 - ii. *Streptococcus fecalis* (Lancefield D Streptococcus) Synonym-Enterococcus
 - iii. Diphtheroids
 - iv. Gonococci (*Neisseria*)

The bacteriology of urinary tract infections appears to be changing. Staphylococcal infections now seem to be more prevalent than *E. coli* infections.

The infection may reach the kidney by the blood stream, by lymphatic spread, or by regurgitation up the ureter. The kidney is permanently damaged and its function impaired. Infections of the skin, bone, intestine, throat, teeth and cervix may act as foci of infection for the urinary tract.

Classification of Urinary Tract Infections.—Cases of urinary tract infection, either pyelonephritis or cystitis, fall into certain natural groups by virtue of the fact that the etiology, pathology, and the prognosis differ greatly. Once a case is diagnosed and it is found to what group it belongs, one can then predict with a high degree of accuracy what the prognosis will be for that particular patient.

The natural groups are:

1. Pyelonephritis only.
2. Groups under 15 years of age.

epithelium of the bladder, ureter, and pelvis of the kidney to cornify (leukoplakia), while under other circumstances such as in ectrophy of the bladder, cystitis, ureteritis, and pyelitis glandularis, the epithelium assumes a glandular form. These changes are precancerous conditions

I PAPILLOMA AND PAPILLARY CARCINOMA

These tumors are usually multiple, appearing as villous growths which are very vascular and bleed easily. They may only involve a small area, or be extensively distributed over the mucosa of the pelvis, ureter, and bladder.

Microscopically the papilloma consists of branching processes containing minute blood vessels, covered with many layers of transitional epithelium. The tumor cells are cuboidal or cylindrical. Round cell infiltration is present in the stroma. The structure is typical and uniform. Though usually benign, it has a tendency to become malignant. Once the tumor invades and infiltrates the submucosa and connective tissue, it becomes a true papillary carcinoma.

Hematuria is an early symptom because of the marked vascularity of the papillary processes.

Metastases occur in the ureter and bladder in over 50% of cases by direct seeding. It is therefore imperative that the entire ureter be removed along with the kidney.

These growths may cause various degrees of hydronephrosis depending upon the site and degree of obstruction.

II NONPAPILLARY TUMORS

1. *Adenocarcinoma* is a large, infiltrating, highly malignant type of tumor which metastasizes rapidly to any part of the body. It is regarded as being a far-advanced papillomatous growth which has lost its papillary character, and has assumed an alveolar or scirrhous appearance.

2. *Squamous cell carcinoma* is a highly malignant tumor which has an insidious onset and runs a rapid and fatal course. It is rare.

Chronic infection, leukoplakia, and calculus are frequently associated with squamous cell carcinoma of the renal pelvis, and are believed to play an important role in its causation.

The squamous pelvic tumor is a greyish, indurated, infiltrating growth which extends over a limited portion of the mucosa. It is large, flat, and ulcerated, but occasionally tends toward a papillary form.

Microscopically the squamous qualities are pronounced. There is usually cornification and pearl formation. These tumors rapidly invade the surrounding structures including the kidney. The prognosis is poor. There has been no 5 year cure reported.

Signs and Symptoms are the same as in other tumors. Hematuria is the most frequent symptom.

Differential Diagnosis.—The conditions to be excluded in the differential diagnosis are renal tuberculosis, renal calculus, pyelonephritis, and tumors of the renal parenchyma.

Treatment is nephroureterectomy, plus the treatment of any seeding present in the bladder.

PRIMARY CARCINOMA OF THE URETER

Ureteral tumors closely resemble bladder tumors in their pathological characteristics.

There are two main groups: (1) Papillary—more common. (2) Nonpapillary.

They occur most commonly in the 4th to 6th decades and are more common in the lower third of the ureter.

Although ureteral tumors are quite similar to bladder tumors pathologically, metastases from ureteral tumors occur earlier and more frequently. This may be due to the insufficient barrier offered by the thin wall of the ureter as compared to the thick wall of the bladder, and to the richer lymphatic drainage of the ureter.

Signs and Symptoms of Tumors of the Ureter.—Hematuria is the commonest symp-

nephritis is its tendency to recur. With each successive attack the interstitial tissue of the kidney progresses to a chronic inflammatory state, so that eventually a chronic pyelonephritis becomes established.

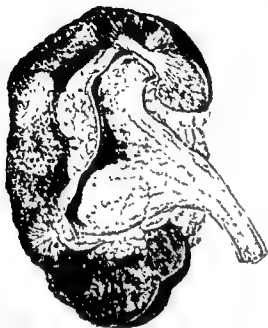


Fig 240.—Chronic pyelonephritis.

CHRONIC

Chronic pyelonephritis is a diffuse infection of the kidney or kidneys which may last for months or years. During this period, acute recurrences of the infection may take place. In the chronic stage the interstitial tissue is filled with lymphocytes, only a few polymorphonuclear leukocytes being present. Colloid casts are present in the tubules which are lined with atrophic epithelium. Periglomerular fibrosis is also present. With each attempt of the kidney to heal, more scar tissue forms, eventually leading to impairment of renal function. The symptoms are those of recurrent infection and pyemia. The treatment is the same as for an acute infection.

Pyelonephritic Atrophy

This process of recurrent exacerbations of infection, followed by recession and the lay-

ing down of scar tissue, may eventually produce a small contracted kidney referred to as *pyelonephritic atrophy*. Renal function may be so impaired by the large amount of scar tissue present that renal failure ensues. The condition may be unilateral or bilateral.

Necrotizing Renal Papillitis

Necrotizing renal papillitis or *necrotizing pyelonephritis* is a severe type of renal infection that is encountered usually in diabetics. Latent infections in the urinary tract may serve as the foci for the development of more severe lesions at a later date. Pathologically the kidney shows multiple small abscesses in the renal pyramid about the level of two-thirds of the way from the papilla to the cortex. As the abscesses coalesce, complete necrosis of the terminal two-thirds of the papilla occurs. The process usually involves all the pyramids of the affected kidney. It may be bilateral.

The onset may be fulminating, in which case the patient suddenly becomes desperately ill, or occasionally the process may be slightly less rapid, although equally violent in effect. High spiking temperature, prostration, and rapid pulse are present without signs or symptoms suggestive of urinary tract involvement. The course is rapidly fatal. A correct diagnosis is rarely made before death.

Pyonephrosis

Pyonephrosis refers to an enlarged kidney containing pus. As any inflammatory condition in the kidney progresses over a prolonged period, the delicately defined contour of the calyces is lost. They become club-shaped, and gradually increase in size at the expense of the cortex which is thinned out. In a well-developed case the outside of the kidney appears lobulated, the lobulations corresponding to the thinned-out soft cortex overlying distended calyces. The products of inflammation (pus) accumulate within the kidney. The kidney eventually becomes a mere pyonephrotic sac (pus sac)

3. Groups complicated by renal calculi
4. Associated with pregnancy.
5. Cystitis only.
- Associated with stricture of the urethra in the female.
7. Associated with overgrowth of the prostate
- Occurring in fractured pelvis, ruptured bladder, and cord bladder

Pyelonephritis

ACUTE

In acute pyelonephritis, one or both kidneys may become involved in the inflammatory process. Pyelitis alone almost never occurs, there being an associated pyelonephritis in 99% of cases. This is evidenced by the finding of an acute inflammatory reaction in the interstitial tissue similar to that found elsewhere in the body. The interstitial tissue and tubules are filled with polymorphonuclear leukocytes.

The infection may initially reach the kidney by

1. The blood stream (hematogenous)
2. The lymphatics
3. Direct ascent up the ureter (urogenous). This may occur in neurogenic bladder associated with atonic, neurogenic ureters (reflex up the ureters).

It is probable that most renal infections are hematogenous.

Symptoms.—The patient usually complains of pain or a sense of fullness in the affected kidney. There is a high spiking temperature, and the patient looks toxic. Bladder symptoms of frequency and urgency are usually present when the bladder is involved.

Diagnosis.—A diagnosis is made upon the symptoms described, the presence of tenderness of the affected kidney to palpation or tenderness in the costovertebral angle, plus the finding of pus in the urine.

Treatment.—The treatment consists of keeping the patient flat in bed with only one

pillow so as to provide better drainage for the kidneys. This is continued throughout each 24-hour period, the patient being allowed to turn on his side to eat, but not allowed to sit up. Fluids are given in large amounts. The appropriate chemotherapeutic agent or antibiotic is given. Large hot linseed poultices are applied to the loin for the relief of pain. Codeine or analgesics may also be given if required. Rarely, a ureteral catheter has to be passed to the renal pelvis and left there for a few days to provide adequate drainage.

Chemotherapy.—There are several chemotherapeutic agents and every effort is made to select the one effective against the organism involved. Any one drug is continued for a period of not longer than 10 days in order to avoid renal irritation. The drugs most commonly used are (1) mandelic acid, (2) sulfonamides; (3) Gantrisin; (4) mandelic acid and Urotropin, (5) Pyridium; (6) arsenicals for resistant staphylococcal infections; (7) antibiotics (penicillin, streptomycin, aureomycin, Chloromycetin, terramycin).

In cases of pyogenic infections of the urinary tract in which a streptomycin-sensitive organism is present, approximately 45% of the cases can be cured by streptomycin. In the presence of urinary tract obstruction, calculi, or foreign body (urethral catheter, suprapubic tube, or ureteral splint), sterilization of streptomycin-sensitive organisms in the urinary tract cannot be effected by the usual doses of streptomycin. Changes in the bacteriological flora of the urine during antibiotic therapy is a common finding and may be an indication for changing to another type of antibiotic.

Sequelae.—With the above regime, the acute infection usually quickly subsides. Resolution may become complete, and healing of the inflammatory process with scar tissue replacement occurs. However, one of the most characteristic features of pyelo-

corpus luteum hormones. This excretion in the urine increases progressively throughout pregnancy.

The incidence of dilatation of the ureter in pregnancy is 52% up to the fifth month of pregnancy, and 92% up to the ninth month. If the incidence of dilatation of the ureter is charted on the graph, it is found that its mean parallels exactly the increased excretion of estrin and corpus luteum hormones during pregnancy.

If one now charts on the graph the incidence of pyelonephritis during pregnancy, it is found that it parallels exactly the incidence of dilatation of the ureter, and both parallel the increased excretion of estrin and corpus luteum hormones in the urine as pregnancy advances.

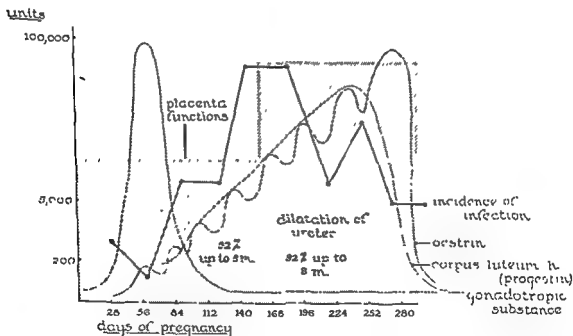


Fig. 242.—Relationship between pyelonephritis of pregnancy, hormone excretion in urine, and dilatation of ureter.

(It has been shown in the Rhesus monkey that if the fetus is removed from the uterus and the placenta left in place, dilatation of the ureter continues. This is what one would expect, since dilatation parallels the excretion of estrin and corpus luteum hormones.)

Cystitis Only

1. Treatment consists of chemotherapy and bladder lavage. The drug of choice will depend upon the organism present.
2. Sitz-baths are of some value.
3. Stricture of the urethra if present will require dilatation.
4. Prostatitis if present will also require treatment.

Cases of Infection Associated with Stricture of the Urethra in the Female

Stricture of the urethra in middle-aged women is a common finding. There is often an associated cystitis and, less frequently a pyelonephritis. The infection disappears promptly after urethral dilatation, bladder lavage, chemotherapy, and estrogen therapy.

The chemotherapeutic drug of choice will depend upon the organism present.

Infections Associated with Overgrowth of Prostate

The bladder becomes infected immediately when it is opened and a tube inserted.

and is almost if not entirely nonfunctional. If the ureter is open, there is purulent drainage from the sac.

The treatment of pyonephrosis is nephrectomy

4. Twenty per cent have a congenital lesion in the urinary tract, e.g., aberrant renal vessel, valves in the urethra, etc.

5. Thirty per cent require a major surgical procedure such as a plastic procedure on the renal pelvis, or a nephrectomy, before a cure can be effected.

The Group Complicated by Renal Calculi

1. The infection cannot be cleared up until the calculus is removed. Only 50% succeed in clearing up their infection completely.

2. Thirty per cent develop recurrence of infection.

3. Forty per cent eventually require nephrectomy.

The Group of Infections Associated with Pregnancy

1. The incidence of infection diminishes with each successive pregnancy.

2. Two-thirds of all the infections occur in the first and second pregnancies.

3. Eighty-five per cent of the patients can be carried to term by conservative treatment

4. Ninety per cent of these can be delivered of living babies

5. Twenty-five per cent are cured during the pregnancy.

6. Ninety per cent are cured within two weeks of emptying the uterus

Endocrinological Aspects of Pyelitis of Pregnancy.—The exact cause of dilatation of the ureter during pregnancy is unknown. Some observers believe it is due to the weight of the gravid uterus, others believe that it is due to hormonal factors.

During pregnancy the gonadotropic hormone reaches a maximum excretion in the urine on the fifty-sixth day, and thereafter rapidly declines. At the third month of pregnancy the placenta takes over the function of producing and secreting estrin and

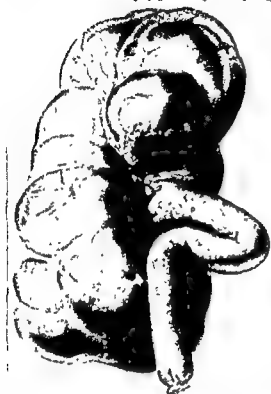


Fig 241—Pyonephrosis

Group of Infections in Children Under 15 Years of Age

Pyogenic urinary tract infections in children show many features in common. The symptoms are more often referred to the gastrointestinal than to the urinary tract.

1. Response to treatment varies directly as the length of time the symptoms existed before treatment is started

2. Twenty-five per cent of these patients have recurrent attacks of pyelonephritis 1 to 10 times.

3. Twenty-five per cent of the cases have an acute upper respiratory infection immediately preceding the urinary tract infection

The carbuncle may rupture into the renal pelvis and empty itself spontaneously via the ureter, or it may rupture into the perinephric space producing a perinephric abscess.

An acute fulminating infection is treated with intensive penicillin therapy. Nephrectomy may be required during the acute stage to save the patient's life, but if possible is deferred. When the acute condition has subsided, nephrectomy will be required if the suppurative process is found to be extensive.

Abacterial Pyuria

Abacterial pyuria is an infection of the urinary tract in which numerous pus cells are found, but in which one fails to recover organisms either on direct smear or on culture. These infections respond very promptly to the intravenous administration of arsenicals (neoarsphenamine, Mapharsen). This has led to the belief that the infection is caused by an unidentified spirochete. The prompt response to arsenical therapy has led others to believe that the infection is caused by Leishman-Donovan bodies.

URINARY CALCULI

Theories of Origin

Theories concerning the etiology of urinary calculi are numerous and conflicting. There is no one constant series of events found in all cases presenting calculus formation.

The etiological factors may be classified as follows:

1. Personal Factors.—

1. Deficiency of vitamin A
2. Dehydration.
3. Prolonged decubitus.
4. Bacterial infections
5. Congenital anomalies causing obstruction.

6. Hyperparathyroidism.

Until recently, *deficiency in vitamin A* was considered to be of great importance in the formation of urinary calculi. Subsequent studies of patients with calculi, however, revealed that only a very few were actually deficient in vitamin A. This theory has therefore been discarded.

Dehydration appears to be of some significance in the formation of urinary calculi. More cases of patients suffering from colic are seen in the hot summer months than during the winter. In World War II, the incidence of calculi in the troops involved

in desert warfare was unusually high. This was thought to be due to dehydration.

It is generally accepted that *prolonged decubitus* tends to increase the incidence of urinary calculi. When a patient is immobilized for a prolonged period, there is increased mobilization of calcium which is derived from the whole skeleton, but especially from the long bones.

There is an increased tendency to stone formation in patients so immobilized. This is considered due in part to increased mobilization of calcium, and partly to the effects of stagnation caused by the prolonged supine position. It is interesting to note that, in this position, the renal pelvis is at the lowest level in the urinary tract in the horizontal plane. This means that urine has to pass upward to a higher level to reach the ureter and flow into the bladder. There is thus a natural tendency to stagnation in the renal pelvis when a patient is kept in the decubitus position for any length of time. This particular factor can be overcome by the use of an oscillating bed or by placing a board frame with a central fulcrum under the standard mattress, so as to permit the patient to alter his position.

Bacterial infection is the main causative factor in many urinary calculi. This is con-

Following a prostatectomy every patient has a cystitis. Its duration will depend upon the intensity of the chemotherapeutic measures directed at elimination of the infecting organisms. If the infection is not cleared up postoperatively, fibrosis of the bladder neck may occur. As a rule it requires two to three months of postoperative care to eradicate the infection. Treatment consists of bladder irrigations once a week and the appropriate chemotherapeutic or antibiotic agent according to the bacteria present.

Infections Occurring in Fractured Pelvis, Ruptured Bladder, and Cord Bladder

Each case is treated as an individual problem according to the principles outlined above.

Perinephric Abscess

Perinephric abscess is an abscess which forms around the kidney and is almost always due to rupture of a cortical abscess. The organism most commonly found in the pus is the staphylococcus, the next most common being *B. coli*. It frequently follows staphylococcal abscesses on the skin or inside the nose.

Symptoms.—The symptoms most commonly found are pain, a sense of fullness in the lumbar region, chills, fever, and prostration.

Physical Signs.—As the abscess increases in size it will spread downward along the psoas muscle and also posteriorly in the loin, causing a bulging with recognizable deformity. The bulging area is tender to palpation, and may be fluctuant.

X-Ray Signs.—The x-ray signs of a perinephric abscess are usually diagnostic. They are:

1. Curvature of the lumbar spine with the convexity away from the abscess
2. Obliteration of the psoas muscle shadow on the affected side.
3. The ribs, or lumbar transverse processes, or both, may be obscured

4. There may be displacement of the colon or fixation of the diaphragm on the same side if the abscess has existed for some time.

5. The kidney may be shown by intravenous pyelograms to be fixed and to lack its normal mobility.

¶ There may be anterior displacement of the kidney.

7. In some cases of perinephric abscess, there is lateral displacement of the kidney with medial displacement of the ureter.

8. Pyelograms may show rotation of one calyx, indicating the presence of an abscess.

Diagnosis is made on the symptoms, physical signs, and x-ray findings.

Prognosis.—The abscess will continue to increase in size until such time as it is incised and drained. If it goes unrecognized, rupture into the pleural cavity or into the peritoneal cavity occurs, either of which may be fatal.

Treatment.—If the abscess is recognized early, it may disappear completely with intensive penicillin therapy. If, however, a recognizable mass is present in the loin, incision and drainage must be carried out. Two or three Penrose drains with wicks are inserted to maintain drainage. Penicillin therapy is given. When the acute condition subsides, the functional status of the kidney is determined and a decision made as to whether or not nephrectomy will be required.

Renal Carbuncle

A renal carbuncle may start by the fusion of several smaller cortical abscesses or by the progressive enlargement of one abscess. As it increases in size it becomes demarcated from the surrounding healthy kidney tissue and its core becomes necrotic. The carbuncle may involve one whole pole of the kidney or even the entire kidney together with perinephric fat.

The symptoms are those of an acute infection, with a high temperature and tenderness to palpation in the kidney region.

normal amount of calcium. This suggests that in the normal kidney there is some mechanism which holds back calcium and acts as a filter. If a calculus is present, this mechanism may become damaged allowing an excess of calcium to escape in the urine. Since the normal exchange of calcium and phosphate ions is across the tubular membrane, it is possible that the disease may begin there.

may also radiate into the testicle or the medial aspect of the thigh on the affected side.

There are three points of narrowing along the course of the ureter. A stone is apt to become lodged at these points of constriction. They are: (1) the uretero-pelvic junction; (2) where the ureter crosses the iliac vessels; (3) at the entrance of the ureter into the bladder.

TABLE XV
CLASSIFICATION OF CALCULI

| | URINE | | CHEMICAL COMPOSITION | MINERALOGICAL NAME |
|--|-------|---------------------|--|--------------------|
| | ACID | ALKALINE | | |
| A. ORGANIC | | | | |
| (1) <i>Uric acid calculi</i> | | | | |
| i. Uric acid | + | | | |
| ii. Urates | | + | | |
| iii Xanthine | + | | | |
| (2) <i>Cystine calculi</i> | + | | | |
| B. INORGANIC | | | | |
| (1) <i>Primary Calcium Calculi</i> | | | | |
| i. Calcium oxalate monohydrate | + | | $\text{CaC}_2\text{O}_4 \cdot \text{H}_2\text{O}$ | Whewellite |
| Calcium oxalate dihydrate | + | | $\text{CaC}_2\text{O}_4 \cdot 2\text{H}_2\text{O}$ | Weddellite |
| ii. Calcium carbonatophosphate | | neutral to alkaline | $\text{Ca}_3(\text{PO}_4)_2 \cdot 2 \text{CaCO}_3$ | Carbonatoapatite |
| iii Calcium phosphate | | neutral to alkaline | $\text{CaHPO}_4 \cdot 2\text{H}_2\text{O}$ | Brushite |
| (2) <i>Magnesium Containing Calculi</i> (May also contain carbonate and phosphate) magnesium ammonium phosphate | | + | $\text{MgNH}_4\text{PO}_4 \cdot 6\text{H}_2\text{O}$ | Struvite |

Pathology

Most calculi are due to obstruction, bacterial infection, or ulceration, either alone or in combination. Some form as plaques on the renal papillae without evidence of any of the above factors.

Symptoms of Renal and Ureteral Calculi

A calculus in the kidney may be large and silent. A stone weighing 432 grams (1 pound) was removed from a patient who had no symptoms referable to the kidney. Small calculi produce attacks of acute renal or ureteral colic as they pass down the ureter. The pain begins in the lumbar region posteriorly, is sudden in onset, colicky in nature, and passes downward, forward, and medially along the course of the ureter. It

A sharp or irregular stone may become embedded in the mucous membrane of the ureter anywhere along its course.

Diagnosis of Renal or Ureteral Calculus

1. History of acute pain in the loin, sudden in onset, colicky in nature, radiating downward, forward, and medially into the bladder, testicle or medial aspect of the thigh on the affected side. The pain may be so severe as to cause collapse.

2. On physical examination, there is splinting of the muscles on the affected side and tenderness to palpation.

3. The urine usually shows scattered red blood cells. It should be remembered that a voided specimen of urine is desirable in these cases, as the passage of a catheter may

firmed by the fact that infected urine is obtained by catheterization from the kidney involved and that certain types of calculi, e.g., calcium phosphate, form only in infected urine. The organism *B. proteus* is the greatest offender and the most difficult to eradicate. Staphylococci are the next commonest organisms which predispose to stone formation. Usually it is impossible to eradicate infection in a kidney in which a calculus is present until the calculus has been removed. Conversely, unless the infection is eradicated, calculi will almost certainly reform. The incidence of recurrence of renal calculi varies from 10% to 40% in different clinics.

Patients with *obstruction*, e.g., aberrant renal vessels at the ureteropelvic junction, stricture, etc., are particularly prone to develop calculi. This is due to the effect of prolonged impairment to drainage from the kidney, resultant stagnation of urine, and increased susceptibility to infection.

Approximately 5% of recurrent urinary calculi are due to *hyperparathyroidism*. Hyperparathyroid disease is discussed in Chapter XII.

II Race and Geographical Distribution.—The white and yellow races are relatively susceptible to the formation of urinary calculi. The Negro race is relatively immune.

There appear to be definite stone-bearing areas in the world. Urinary calculi are much more common in India and China than in other countries. Just what effect soil and climate have on the formation of calculi is not known.

III. Theories of Physical Factors Associated with Stone Formation.—Considerable speculation regarding the origin of urinary calculi was based on the results of chemical analysis. In turn, a theory was developed in which the causes were attributed to various physical factors, such as:

(A) *The presence of stone-forming substances in the urine.* Every specimen of urine contains stone-forming elements, e.g.,

uric acid and calcium oxalate. The solubility of these substances depends upon:

(i) The pH of the urine. At pH5 pure uric acid is present to the point of saturation. At pH7, the urine is supersaturated with calcium oxalate.

(ii) The presence of other electrolytes in the urine. The solubility of a sparingly soluble electrolyte is increased by the presence of other more soluble electrolytes, e.g., the solubility of uric acid is increased by the presence of sodium phosphate.

(iii) The presence of certain nonelectrolytes. The solubility of a salt in a solution may be greatly increased by the presence of another salt in the same solution, e.g., the solubility of calcium oxalate in water is doubled by adding urea.

(B) *The protective action of urinary colloids.* The urinary colloids consist of mucin, nucleic acid, chondroitin sulphuric acid and a complex nitrogen-containing carbohydrate. They are suspended in solution and their precipitation may lead to the formation of the nucleus of a future calculus. The nucleus of the developing calculus will continue to grow provided:

- (i) it is retained within the urinary tract,
- (ii) the urine continues to supply an excess of stone-forming salts.

This theory advocated by Swift Joly and others has considerable appeal. It tends to explain the physical factors concerned, but is totally unable to explain many of the other known factors in calculus formation. It is perhaps much closer to the truth to say that the exact cause of calculus formation is not known in all cases, although many of the contributing factors have been recognized.

If a catheter is passed up each ureter to the renal pelvis and specimens of urine are obtained from patients with renal calculi, in whom hypercalcaemia is present, urine from the kidney containing the calculi is found to have an excess of calcium, whereas urine from the other kidney is found to contain a

normal amount of calcium. This suggests that in the normal kidney there is some mechanism which holds back calcium and acts as a filter. If a calculus is present, this mechanism may become damaged allowing an excess of calcium to escape in the urine. Since the normal exchange of calcium and phosphate ions is across the tubular membrane, it is possible that the disease may begin there.

may also radiate into the testicle or the medial aspect of the thigh on the affected side.

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2. On physical examination, there is splinting of the muscles on the affected side and tenderness to palpation.

3. The urine usually shows scattered red blood cells. It should be remembered that a voided specimen of urine is desirable in these cases, as the passage of a catheter may

in itself be sufficiently traumatic to produce scattered red blood cells in the urine

4 A plain x-ray plate of the kidney, ureter and bladder area will reveal the site, size, and number of calculi present if they are nonopaque. Approximately 5% are nonopaque



Fig 243—X-ray of abdomen showing large stag-horn calculus in the left kidney

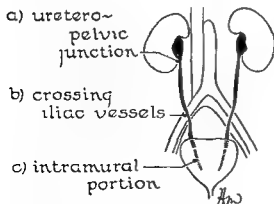


Fig 244—The three points of narrowing of the ureter.

5. Cystoscopic examination and ureteral catheterization is performed. X-rays taken in the anteroposterior and oblique diameters with the catheter in situ will show conclusively whether or not the calculus is in the ureter. If a calculus is present in the ureter, the opacity seen on x-ray will remain in contact with the catheter in both the anteroposterior and oblique projections. However, if the opacity is seen to lie one-half inch or so away from the catheter in either projection it can be said with certainty that the opacity is not due to a calculus in the ureter, regardless of the other symptoms present. In the latter case the shadow may be due to a calcified gland or phlebolith.

Treatment

1. Emergency relief of the colic by giving morphine gr. $\frac{1}{4}$ and atropine gr. $\frac{1}{150}$
2. Prevention—try to correct the underlying condition if possible
3. Surgical—the particular type of surgical procedure to be employed depends upon the conditions present and may be any of the following

I Operations on the Kidney.—

1 Remove the calculi—x-ray at the time of operation to make certain all the calculi are removed.

(i) Pylotomy—i.e., open the pelvis of the kidney and remove the calculus

(ii) Nephrolithotomy—i.e., approach the stone through the substance of the kidney

2. Remove obstructions at the time of the pylotomy or nephrolithotomy, e.g., if ureteropelvic obstruction is present do a plastic procedure at the site of obstruction.

3 Nephrectomy—if the disease is advanced

II Operations on the Ureter.—

1. The ureteral orifice and ureter may be dilated with bougies.

2. Or be cut on the affected side with cystoscopic scissors or a cautery wire.

3. If the calculus is low in the ureter and not too large, a stone basket may be passed

and the calculus removed. Serious trauma to the ureter may be caused by rough handling of a stone basket. An edematous seminiferous ureter may be perforated, the infection thereby being spread outside the ureter into the retroperitoneal plane with the production of a perinephric abscess.

4. The ureter may be incised over the calculus and the calculus removed.

Dietary:—

1. Vitamin A is said to be of some value, but this is doubtful.

2. Cystine stones can be prevented and perhaps even reabsorbed by

- (i) reducing proteins in the food to a minimum,

- (ii) by giving alkalis to keep the urine constantly alkaline.

3. Attempts have been made to dissolve calcium and phosphate calculi by using buffered citric acid solutions as irrigations (Solutions G. and M.) In our experience these have not been successful.

4. Some authors believe that the presence of citric acid in the urine increases the solubility of the calcium salts. Estrogenic hormone is given to increase the excretion of citric acid in the urine, thereby increasing the solubility of calcium. Aluminum hydroxide gel is given at the same time, to increase the elimination of phosphates by the bowel, and diminish the amount available to the kidney.

GENITOURINARY TUBERCULOSIS

Genitourinary tuberculosis may affect the kidney, prostate, seminal vesicles, epididymis, Fallopian tubes or bladder. It may be limited to any one of these organs, with the exception of the bladder. Its most characteristic feature is its marked tendency to spread and this plays the predominant role in deciding the treatment.

It must also be borne in mind that tuberculosis is a generalized infection and that urogenital tuberculosis is only a local manifestation. Such infection is usually secondary to a primary focus in the lungs, intestinal tract, tonsils or bones. The primary focus can be found in approximately 80% of cases. In searching for a primary focus, one should also bear in mind that an early pulmonary lesion may not be detectable by radiological means until six months after its onset.

Age.—Genitourinary tuberculosis is rare under the age of 10 years and, if present at this age, is almost always of the acute fulminating type. Seventy per cent of cases occur between 20 and 40 years of age.

Incidence.—The incidence of genitourinary tuberculosis is approximately 1 to 2%

in routine autopsies; 5% in autopsies on tuberculous patients dying of other diseases; 10% in patients dying of tuberculosis.

The incidence of the bovine type in genitourinary tuberculosis is exceedingly low.

In cases of genitourinary tuberculosis, the prostate, seminal vesicles or epididymis are involved in 65% to 75% of the cases. Primary tuberculosis of the prostate gland alone is rare.

Renal Tuberculosis

Renal tuberculosis is the result of the introduction, implantation, and germination of the tubercle bacillus in the renal bed. The extent of the lesion depends upon the virulence of the bacillus and the resistance of the host.

There are three main types of lesion in the kidney:

1. **Acute miliary**—fulminating bilateral renal lesions which are part of a generalized miliary tuberculosis.

2. **Chronic form**—in which there is abscess formation with caseation.

This is referred to as surgical renal tuberculosis

3 Toxic tuberculous nephritis—which is a renal manifestation of grave systemic tuberculosis, without definite involvement of the urinary organs. It is exceedingly rare.

Etiology.—Renal tuberculosis is always hematogenous in origin, the primary focus being in the lungs, tonsils, intestinal tract or bone. The infection in the primary area progresses until the tubercle bacilli finally invade the blood stream. If the invasion of the blood stream is massive, general miliary tuberculosis results. If the organisms enter the blood stream in showers, the phagocytic cells of the body may overcome them. If the patient's general and local resistance is sufficient, the secondary lesion in the kidney may heal; otherwise it progresses with eventual destruction of the kidney. Renal, and other secondary lesions in the prostate, seminal vesicles, and epididymes may all originate from the same primary focus in the lungs, tonsils or intestinal tract. Conversely, the renal lesion may be derived from infection in the prostate, seminal vesicles, or epididymis, and carried to the kidney by the blood stream. It is the consensus that renal tuberculosis is blood borne, and that tuberculous infection ascending the ureter or the lymphatics does not occur.

Pathogenesis.—It is now generally accepted that a bacillus-laden embolus is carried into the kidney by the blood stream and lodges in a capillary tuft. The glomerular focus may be walled off or a few bacilli may reach the capsular space and multiply while being washed slowly along the proximal convoluted tubule until they reach a favorable soil for growth in the medullary loop. The bacilli may, however, reach the medulla by way of the fine efferent glomerular capillaries. The final method of infection is by spread of cortical lesions to the medulla by the lymphatics.

Opinions differ as to whether the initial lesions in the kidney are predominantly cortical or medullary. Medler, who did 100,

000 sections on 44 separate tuberculous kidneys, found 367 definite tuberculous lesions of which 75% were in the cortex, 11% in the medulla, and 14% were corticomedullary.

The initial glomerular lesion may heal, continue to grow slowly, or rarely develop into the main lesion. The medullary lesion usually grows relatively fast. Such a tubercle will spread, and finally open into a renal tubule or papilla, discharging its contents into the renal pelvis. If the medullary lesion is near a calyx, it will give rise to early symptoms. If it is deep in the parenchyma there may be extensive damage before there are any clinical signs. Healing of this type of lesion is rare.

The Question of Initial Bilateral Involvement.—Of the many disputed points concerning renal tuberculosis none has been more discussed than the question as to whether, at the outset, one or both kidneys are involved. Many investigators believe the implantation is bilateral, but that the disease develops in only one kidney.

The Excretion of Tubercle Bacilli in the Urine.—The presence of tubercle bacilli in the urine represents a break in the integrity of the renal mucous membrane and is regarded as the essential evidence of disease within the renal parenchyma.

Secondary infection occurs frequently. In urine obtained from a tuberculous patient organisms other than tubercle bacilli, e.g., *E. coli*, staphylococci, are found in 20% of cases. It is therefore important to exclude tuberculosis in all cases of persistent urinary infection.

Symptoms of Renal Tuberculosis.—

- 1 Frequency, day and night, is present in 75% of the cases
- 2 Painful urination is often present
- 3 Hematuria—microscopic or macroscopic in 50% of the cases

- (1) Terminal bleeding is common
- (ii) Smoky urine.

(iii) Microscopic red blood cells

(iv) Gross hematuria is rare

1. Dribbling is usually a sign of pronounced vesical involvement.

Diagnosis.—

1. History.

2. The recovery of tubercle bacilli from the urine is the essential evidence of active disease within the renal parenchyma (i) by direct smear, (ii) positive urine cultures, (iii) guinea pig inoculation of urine from each kidney

3 Retrograde pyelograms demonstrating alterations in the renal architecture

Treatment of Renal Tuberculosis.—The incidence of bilateral renal tuberculosis will vary depending upon the promptness with which observation and urological investigation are undertaken. It is bilateral in 20% to 45% of cases. It is of the greatest value to know the patient's ability to fight his



Fig 245 Pyelogram showing deformity caused by renal tuberculosis

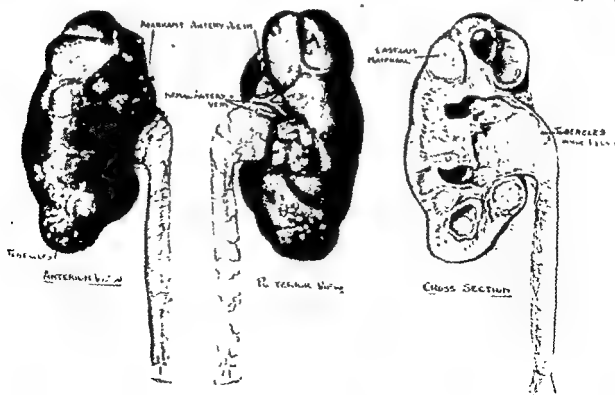


Fig 246.—Gross specimen of renal tuberculosis

disease, the pathological type of the disease, and the prognosis insofar as the pulmonary lesion is concerned

The use of streptomycin with para-aminosalicylic acid has considerably altered the technique of treating renal tuberculosis. Experience with streptomycin, or streptomycin and para-aminosalicylic acid would indicate that at the end of therapy, there is clinical improvement in some, and marked improvement in other cases. In bilateral renal tuberculosis it is usually advisable to remove the more severely infected kidney giving streptomycin and para-aminosalicylic acid concurrently. The chemotherapy is started one week preoperatively and continued for one to six months postoperatively. This frequently results in healing of the lesion in the less involved kidney.

In cases of renal tuberculosis, the normal-appearing ureter will show microscopic tuberculous lesions in 30% of cases. It is for this reason that many surgeons recommend nephroureterectomy for renal tuberculosis, even though the ureter appears normal at the time of operation.

Summary of Treatment.—(Table XVI). All cases of genitourinary tuberculosis, especially after surgery:

1. Complete bed rest.
2. Sanatorium treatment, or the equivalent at home for a period of 6 months.

The severity of the symptoms should be a strong factor in determining treatment. One does not like to do a bilateral cutaneous transplant because its care is difficult for the patient.

TABLE XVI
TREATMENT OF RENAL TUBERCULOSIS

| | UNILATERAL DISEASE | BILATERAL DISEASE |
|--------------------|--|--|
| Medical Treatment | Streptomycin and para-aminosalicylic acid. | Streptomycin and para-aminosalicylic acid |
| Surgical Treatment | Nephrectomy or nephroureterectomy to be preceded and followed by streptomycin and para-aminosalicylic acid | 1. Remove the more diseased kidney 2. Bilateral cutaneous ureteral transplant in advanced bilateral disease. To be preceded and followed by streptomycin and para-aminosalicylic acid |

Tuberculous epididymitis responds very slowly, if at all, to streptomycin therapy. This lack of response may be due to failure to obtain an adequate drug concentration in the tissue. Tuberculous scrotal sinuses respond promptly to streptomycin therapy.

Streptomycin is not used in patients with a small contracted tuberculous bladder as this will achieve only slight improvement of the clinical symptoms, but will not alter the over-all picture.

Institution of operative measures at the proper time for chronic renal tuberculosis rarely causes a flare up of the associated pulmonary disease. Surgery should be delayed whenever possible until the pulmonary lesion has become quiescent.

Tuberculosis of the Bladder

Tuberculosis of the bladder is secondary to tuberculosis of the kidneys or seminal tract in the male. In the female it may be secondary to tuberculosis of the pelvic organs. In the majority of cases the primary focus is in the kidney. The ureteral orifice on the affected side is usually involved.

Involvement of the bladder, secondary to a tuberculous focus in the prostate or urethra occurs by direct mucosal extension. In such cases the trigone is practically always the point of first attack. In most cases, however, the bladder involvement is due to the planting of tubercle bacilli from infected urine directly on the vesical mucosa, the resistance of which has been lowered by the

continuous irritation of infected urine. This leads to inflammation, edema, ulceration and finally fibrosis of the bladder wall. The bladder becomes contracted and its capacity greatly reduced.

Symptoms.—

1. Intense frequency.
2. Painful urination; the pain occurring at the end of micturition.
3. Slight, intermittent hematuria

Diagnosis.—

1. Appearance of the bladder at cystoscopy, small tuberculous ulcers.

2 The recovery of tubercle bacilli from the urine.

Prognosis.—If the source of the vesical infection is removed (usually a tuberculous kidney), the bladder condition often heals rapidly. If, however, the original source of the disease is not eliminated, it is practically impossible to cure, or even materially relieve, a tuberculous cystitis. When treatment is completed, sanatorium care should be instituted.

Tuberculosis of the Prostate

Tuberculosis of the prostate alone is very rare. It is usually part of a progressive infection that is extending throughout the genital or urogenital system, being present in about 70% of cases of urogenital tuberculosis. In most instances tuberculosis of the prostate occurs by direct extension from the seminal vesicles. Urogenital tuberculosis tends to spread from one portion of the urinary tract to another.

Symptoms of Tuberculosis of the Prostate.—

1. None at all
2. Frequency; dysuria, hematuria; pyuria.

Diagnosis.—

1. Rectal examination—the prostate feels firm, irregular and nodular.
2. Examination of the prostatic fluid for tubercle bacilli.

3. A plain x-ray of the pelvis may show areas of calcification in the prostate.

Treatment.—

1 Total perineal prostatectomy gives poor results. Chronic sinuses may result.

2 Sanatorium rest or the equivalent thereof often gives relief of symptoms, although the course of the disease is essentially unaltered.

3 Antibiotics.

Tuberculosis of the Epididymis and Testicle

Autopsy records show that tuberculous lesions may be limited to the epididynes, seminal vesicles, or prostate, but that such limitation is rare. Clinically the majority of cases exhibit multiple lesions. The infection in the epididymis usually begins in the globus minor, and gradually extends throughout the epididymis and into the vas. The tuberculous process in the epididymis may assume such extensive proportions that the testicle becomes a small compressed organ.

The disease sometimes appears to be bilateral from the onset. When tuberculosis infects only one epididymis the opposite epididymis is likely to become infected sooner or later. For this reason it is advisable to do a vasectomy on the unaffected side.

Tuberculosis of the testicle is very rare. It is usually secondary to tuberculosis of the epididymis. The entire testicle may become a hard mass by gradual extension of the disease. The scrotum becomes adherent to the mass; the process finally ulcerates through the skin to form a chronic fistula discharging caseous material. The function of the affected organ is destroyed and, should the process become bilateral, complete sterility is inevitable.

Sinuses on the posterior surface of the scrotum are from the epididymis. Those on the anterior surface are from the testicle. A persistent scrotal sinus of months' or even years' duration is frequently the reason the patient seeks medical advice.

Diagnosis of Tuberculosis of the Epididymis.—

1 History of trauma is present in many cases, frequently it is this that draws the patient's attention to the fact that there is ■ swelling present in the scrotum.

2 Loss of weight and general malaise are usually present

3 Bladder symptoms may be present.

4 Fever is rare.

5 Pain and tenderness in the epididymis are mild and intermittent.

6 History of exacerbations and remissions of the swelling

7. On palpation the epididymis is found to be swollen, nodular, and irregular in contour.

8 A persistent draining sinus may be present

Differential Diagnosis.—

1. Tuberculous epididymitis.

2 Inflammation due to other causes.

Prognosis and Treatment.—The high morbidity of genital tuberculosis, even when properly treated, is not generally appreciated. The disease is progressive and fairly rapid in its evolution. Prior to the use of antibiotics the ultimate mortality varied from 27% to 60% The patient must be examined yearly for ten years.

Treatment.—

1 Epididymectomy—with transplantation of the vas to the skin of the groin

2 Six months' sanatorium treatment or the equivalent at home.

3. Antibiotics

TUMORS OF THE ADRENAL GLAND

Cortical Tumors

Tumors of the adrenal cortex may be (1) due to simple hyperplasia, these are benign and may be diffuse or nodular; (2) adenomas which are benign; and (3) carcinomas or hypernephromas.

Symptoms.—The symptoms may vary greatly depending upon the type of tumor and its location. Hyperactivity produces symptoms of sexual precocity and pseudohermaphroditism, e.g., infant Hercules, and bearded lady. Hypoactivity produces infantilism and asthenia in early life. In adult life it produces lack of sexual desire in the female, and in the male, impotence, obesity, and senilism. The presence of an adrenal cortical tumor may be suspected because of

- (i) the endocrine symptoms,
- (ii) the presence of an abdominal mass,
- (iii) the finding of metastatic masses, or
- (iv) the presence of hypertension

Pathology of Cortical Tumors—

1. *Simple Hyperplasia* occurs fairly frequently in adults, and may produce one or

more nodules or diffuse enlargement. Nodules, if present, may vary from the size of a pinhead to the size of a pea and, like adenomas, are benign. Sometimes the cortex shows diffuse hyperplasia with great enlargement of the adrenal gland. This form gives rise to a clinical picture similar to that produced by a cortical neoplasm

2 *Adenomas* are the most frequent tumors of the adrenal cortex. They vary in size from 1 to 2 cm, are mottled brown and rarely give clinical evidence of their presence. They are often encapsulated, usually remain small, are often bilateral, and are often only discovered at autopsy

The distinguishing characteristics of the adenomas are the definite cortical cells, the tendency to lipoid degeneration, a marked capillary network, pigment deposits, and the arrangement of cells into cords and bundles. Larger ones are distinguished by areas of vascularization. These features distinguish adenomas from the localized nodules of hyperplasia. The absence of tubular or papil-

lary structures distinguishes them from hypernephroma of the kidney.

3. *Carcinomas* of the adrenal cortex are rare. They are soft growths of a yellowish color and varied consistency. They have a tendency to develop hemorrhagic and necrotic areas followed by cystic degeneration. These tumors metastasize early and widely through the lymphatics to the venous system, most frequently to the liver and lungs, but rarely to the bones. Occasionally they metastasize to the opposite adrenal gland. Invasion of the kidney, renal vein, and vena cava by direct extension is common.

Tumors arising from adrenal rests occasionally occur in the liver, kidney, testes, broad ligaments, and retroperitoneal tissues.

Treatment.—Surgical removal.

Medullary Tumors

The function of the adrenal medulla is related to the sympathetic system and the organs over which the sympathetic system exerts an effect, e.g., the heart and blood pressure.

cell, namely, the sympathetic formative cells (sympathogonia). These cells differentiate into the sympathoblast (nervous type) and the pheochromocyte (endocrine type).

Tumors of Adrenal Medulla

1. *Neuroblastoma*.—These tumors develop from the primitive undifferentiated neuroblast. They grow rapidly and are highly malignant. They extend early to both sides of the spine, producing extensive metastases throughout the body. The metastases consist of soft tumor masses similar to the primary growth.

The growth is usually reddish or reddish brown in color, well encapsulated, and is often firm and nodular in consistency, though it may be soft and spongy. It usually depresses the kidney on the affected side, but does not invade it.

On microscopic section the neuroblastoma shows cells of the sympathetic system in various stages of development (sympathogonioma, sympathoblasts, or neuroblasts). The cell most commonly seen is larger than a

TABLE XVII
ADRENAL MEDULLARY TUMORS

| EMBRYOLOGICAL CELL | NORMAL DEVELOPMENT | TYPE OF TUMOR |
|----------------------------|---|---|
| Sympathetic formative cell | 1. <i>Nervous type</i> sympathoblast ganglion cell | <i>Nonhormonal type</i> sympathoblastoma ganglioneuromata (benign) neuroblastomata (malignant) |
| | 2. <i>Endocrine type</i> pheochromoblast pheochromocyte | <i>Hormonal type</i> pheochromocytoma—rare. |

TABLE XVIII

| TUMOR OF RIGHT ADRENAL GLAND | TUMOR OF LEFT ADRENAL GLAND |
|---|---|
| Pepper type of metastases—to { liver and retroperitoneal lymph nodes (Right lymphatics empty directly into the portal system, and the right adrenal gland lies in direct contact with the liver) | Hutchinson type of metastases to { skull and orbit (Left lymphatics are connected with lumbar glands and pass downward to groin and aortic glands, and upward to the deep cervical glands and the skull) |

The tumors of the medullary portion of the adrenal gland may be divided clinically into those which produce hormonal symptoms, and those which do not. Embryologically both types of tumor arise from the same

lymphocyte with a minute amount of slightly basophilic and finely granular cytoplasm. This is a rapidly multiplying, highly embryonic, undifferentiated round cell. The cells are often embedded in pseudorosette forma-

tion, in an intercellular substance consisting of a very fine network of minute fibrils. Hemorrhage, often massive in type, and necrosis are very common findings. Direct extension of the growth to neighboring viscera frequently takes place, sometimes before distant metastases are observable.

Patients with the Pepper type of metastases usually have a tumor in the right adrenal gland. Patients with the Hutchison type of metastases may have a tumor on either side. These syndromes may, however, occur with other tumors of the adrenal gland and occasionally of the kidney.

2 Ganglioneuroma.—This type of tumor is very rare. It develops from the mature sympathetic ganglion cell. It may be discovered as a small growth at autopsy, or may grow to a large size. It is usually single but may be multiple and is almost always benign. The structure shows a preponderance of medullated and nonmedullated nerve fibers, among which groups of more or less well-formed proliferating ganglion cells are found. If malignancy is present its degree depends on the relative proportion of mature and immature cells.

3 Pheochromocytoma.—(*Synonyms; paraganglioma; chromaffinoma.*) These are rare, small, reddish tumors which may occa-

sionally reach a large size. They are usually benign.

Symptoms of Pheochromocytoma are those of medullary hyperactivity, i.e., paroxysmal hypertension. These cases demonstrate the exaggerated response of the body to the introduction of large amounts of epinephrine into the blood. There is pounding headache, nausea, dyspnea, orthopnea, palpitation, blanching of the skin, paraesthesias, abdominal cramps, vomiting, precordial throbbing and marked weakness. An attack lasts from a few minutes to 36 hours and may send the patient into shock. It terminates with flushing of the blanched areas, marked perspiration and prostration.

Death may occur during an attack from shock, pulmonary edema, left cardiac failure, coronary disease, or cerebral accident.

If left untreated arteriolar sclerosis eventually results from unchecked vascular tension and the hypertension becomes fixed.

The attacks may occur spontaneously or they may be induced by any mechanism which calls forth an epinephrine discharge (pressor response).

Treatment of Adrenal Tumors.—If the tumor is malignant, the ultimate prognosis is hopeless. Surgical removal of the adrenal gland is the treatment to be carried out in suitable cases.

THE BLADDER

Anatomy.—The bladder is a reservoir for urine. It is a musculo-membranous sac situated in the bony pelvis behind the pubis and in front of the rectum in the male. In the female it is separated from the rectum by the cervix uteri and the vagina.

When empty, the bladder is pyriform in shape, with the apex below. The ureter of each side is connected to each of the upper angles of the triangle, and the urethra is connected below. The urachus (obliterated allantoic duct) connects the bladder to the umbilicus. The superior or upper surface of the bladder is covered with peritoneum.

The normal bladder capacity is approximately 500 c.c. of fluid.

The interior of the bladder is lined with folds and shows on the floor the vesical trigone, a pale-colored triangular space, marked at its upper and outer angles by the entry of the ureter on each side (ureteral orifice) and extending below to the opening of the urethra. The interureteric ridge runs transversely between the two ureteral orifices.

Blood Supply.—The arteries of the bladder are the superior vesical, middle vesical, and inferior vesical in the male, with addi-

tional branches from the uterine and vaginal arteries in the female. These are all derived from the anterior trunk of the internal iliac artery.

The veins form a plexus around the neck, sides, and base of the bladder, and terminate in the internal iliac veins.

The lymphatics terminate in the internal iliac glands.

divides the cloaca into an anterior or urogenital part, and a posterior or intestinal part.

Nerve Supply. The nerve supply of the bladder, ureter, and urethra is of three sources—sympathetic, parasympathetic, and somatic. The sympathetic fibers which innervate the bladder are derived from T11, 12 and L1-2, and come together in a strand

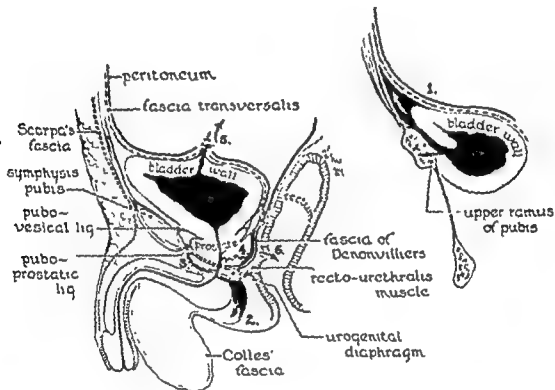


Fig 247—Anatomical relations of the urinary bladder, urethra, and rectum, showing sites of injury.

Developmental Embryology

The rudimentary alimentary canal ends below in a blind pouch known as the cloaca, which has a prolongation known as the allantoic diverticulum which later becomes the urachus. The mesonephric duct which later becomes the vas deferens opens into the anterior part of the cloaca. The ureters develop as an outgrowth of this duct. In time this common duct is absorbed into the wall of the bladder and prostatic urethra, with the result that the vas and the ureter eventually develop separate openings. A urorectal septum then forms which

called the *presacral nerve*. Lateral roots connect this to the 1st and 2nd lumbar prevertebral sympathetic ganglia. A middle root from the preaortic nervous plexus also joins it. This network provides intercommunication between the celiac, semilunar, and aorticorenal ganglia. At the level of the promontory of the sacrum the presacral nerve contains both afferent and efferent fibers.

The *parasympathetic* fibers arise from the sacral segments 2, 3, 4 and run through the hypogastric plexus. The proximal portion of the hypogastric plexus forms a nerve group

which is motor to the bladder and causes contraction. The middle portion supplies the urethra and genitalia, the distal portion innervates the bowel

The *somatic fibers* from Sacra 3 and 4, pass in the *puddendal nerves* to innervate the striated muscle around the urethra and the external sphincter of the bladder. The pudendal nerve also contains sensory fibers from the posterior urethra

Physiology of Micturition.—For many years it has been accepted that there is reciprocal innervation of the bladder, i.e., that the parasympathetic contracts the detrusor muscle and inhibits the internal sphincter, conversely, that the sympathetic contracts the internal sphincter and relaxes the detrusor. This view is no longer held. Anatomically there is no true sphincter at the bladder neck. The detrusor muscle of the bladder, i.e., the longitudinal layer, is continuous with the bladder neck, so that what has been called the internal sphincter is really simply the edge of the detrusor muscle. The circular muscular layer of the bladder is responsible for the maintenance of adequate intravesical pressure. The sympathetic nerves play no part in micturition. They carry pain and temperature sensations only.

The act of micturition is essentially a spinal reflex. Distention of the bladder causes a desire to void. This stretch reflex is carried by the afferent fibers in the parasympathetic nerves to the sacral spinal cord and up the cord to the pons, subcortical, and cortical areas of the brain where involuntary and voluntary control, respectively, are normally situated. With the removal of voluntary control, the act of micturition is initiated by a contraction of the vesical muscle (i.e., detrusor or longitudinal muscle) via the *parasympathetics*. Once this contraction is fairly well established, the internal orifice of the bladder (bladder neck, "internal sphincter") opens, and the external sphincter (supplied by the pudendal nerve, i.e., somatic) relaxes.

The Neurogenic Bladder

Immediately following serious trauma to the spinal cord in which there is spinal transection, a condition known as "spinal shock" ensues. This is a state of depression of the spinal reflexes in the segments caudal to the lesion. It is generally transient in character

The actual mechanics of this condition are unknown, but it causes paralysis with loss of reflexes below the level of the lesion. The bladder is immediately affected as shown by:

- 1 Atonicity of the bladder wall.
- 2 Absence of reflex contraction of the detrusor .
- 3 Failure of sensory impulses to the brain and motor impulses from it.

The bladder distends to a large size, overflow incontinence occurs in 36 hours.

The atonic bladder noted immediately after injury in the state known as *spinal shock* is the first of three merging but fairly well-defined stages of spontaneous recovery

First Stage may last one day to 18 months. Overflow incontinence is the only type of urination to be expected. Cystometric studies during this stage show a large bladder capacity with low pressure, and without evidence of reflex detrusor activity, i.e., atonic neurogenic bladder

Second Stage is a variable period of time from days to months after the injury; a return of reflexes caudal to the injured segment usually takes place. It is manifested in the cystometrogram by higher intravesical pressure. Reflex contractions of the bladder wall occur, but they are of insufficient strength or duration to produce efficient emptying. The clinical picture is that of periodic overflow incontinence, i.e., autonomous neurogenic bladder

Third Stage.—Progression to a third and final stage of recovery depends upon the extent of the injury and upon whether (i) transection has been partial or complete;

(ii) level of injury; (iii) general condition of the patient; (iv) degree of infection in the bladder.

Treatment of Bladder in Spinal Cord Injuries

Immediate Care.—

1. Repeated catheterization at intervals is condemned because severe infections ensue.

2. Indwelling urethral catheter, either with or without tidal drainage, is not to be recommended, except in cases where fairly prompt recovery is anticipated. A peri-urethral abscess is a frequent complication.

3. Suprapubic cystotomy is usually the treatment of choice. With the bladder draining well through a large suprapubic tube, infection can be controlled. The cystotomy should be as high as possible. Some objections to this type of immediate treatment have been raised, the claim being made that with prolonged drainage, fibrosis of the bladder occurs and the bladder capacity is permanently reduced. This, however, has never been proved.

4. Urinary antiseptics should be used commencing at the time of the injury.

Late Care.—

1. Continue with mild urinary antiseptics throughout the period of treatment. The intention is to obtain a bacteriostatic rather than a bactericidal effect.

2. Introduce methods to prevent calculus formation, i.e., move the patient frequently from side to side, get patient up if possible, otherwise use a tilting board under the mattress.

3. Keep the bladder clean by repeated irrigations, using either a bulb syringe or tidal irrigator apparatus. Solutions of boric acid, sulfonamides or buffered citric acid solution, G, M, or F, may be used for irrigation.

4. Remove any calculi that form in either the bladder or kidneys.

5. When recovery appears to be taking place, as evidenced by a return of reflexes

caudal to the injured segment and the showing of a higher intravesical pressure on the cytometrogram, the suprapubic tube can be removed, and the bladder allowed to close. An indwelling urethral catheter may be of assistance at this time. Rarely is a secondary closure of a suprapubic wound required.

Final Stage.—Recovery to the final stage is often surprisingly satisfactory. If the lesion is one of the cauda equina, fairly good control of the bladder can be developed.

In many cases, fibrosis of the bladder neck occurs with resultant hypertrophy of the adjacent muscle. An hourglass type of bladder neck develops. This may be relieved by a transurethral resection in which successive pieces of tissue are removed from around the bladder neck. It is desirable to eliminate residual urine. Repeated resections may be necessary to accomplish this.

Many patients in spite of careful treatment continue to have a residual urine of 1 to 2 ounces. In these patients a catheter should be passed once a week, and the bladder thoroughly irrigated, as stagnant residual urine is prone to become infected and form calculi.

(See Infections of the Bladder, pp. 553-559.)

(See Acute Urinary Retention, pp. 581-585.)

Rupture of the Urinary Bladder

Rupture of the urinary bladder is usually due to external violence when the bladder is distended. The bladder when contracted often escapes injury even in violent crushing accidents. Rupture of the bladder is more common in males.

Etiology.—The types of trauma causing rupture are:

1. Crushing injuries.

2. Falls.

3. Blows and kicks.

4. Manual emptying of the bladder.

The tear is generally on the anterior wall, and it is caused by fragments of the frac-

which is motor to the bladder and causes contraction. The middle portion supplies the urethra and genitalia; the distal portion innervates the bowel.

The *somatic fibers* from Sacra 3 and 4, pass in the *puddendal nerves* to innervate the striated muscle around the urethra and the external sphincter of the bladder. The pudendal nerve also contains sensory fibers from the posterior urethra.

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Third Stage—Progression to a third and final stage of recovery depends upon the extent of the injury and upon whether (i) transection has been partial or complete;

Prognosis.—The prognosis is good if the injury is recognized early and the bladder is drained

Tumors of the Bladder

Classification

- I. Papillary.
papilloma.
papillary carcinoma.

2. Malignant
sarcoma
rhabdomyosarcoma

Theories of Origin.—The cause of cancer is unknown. There are several theories as to the cause of bladder tumors:

1. Virus infections.
2. Chemical irritation.

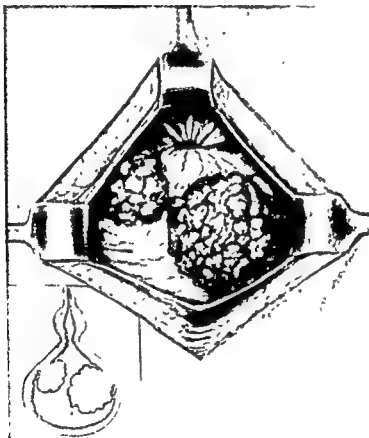


Fig 249.—Multiple papillomas of bladder with malignant invasion of base.

- II. Solid
transitional cell carcinoma
squamous cell carcinoma.
adenocarcinoma (mucus secreting).
anaplastic carcinoma.

3. Occupational, e.g., bladder tumors in aniline dye workers and the scrotal cancers of chimney sweeps.

4. The influence of the constant flow of urine

III Mesoblastic tumors

- 1 Benign growths
fibroma
myoma
myxoma
angioma

PAPILLOMA—PAPILLOMATOSIS

Papillomas of the bladder may be single or multiple. When several small growths occur together or in rapid sequence, the condition is termed papillomatosis.

tured pelvis piercing the bladder. The second common site is on the posterosuperior wall. In every case of fractured pelvis, rupture of the bladder should be excluded

Pathology.—The bladder tear may be either *intrapertoneal* (40%) or *extraperitoneal* (60%). An intraperitoneal rupture occurs when the posterosuperior wall of the bladder is torn. Blood and urine extravasate into the peritoneal cavity causing a peritonitis with abdominal distention, tenderness, rigidity, fever, and rapid pulse. Extraperitoneal rupture occurs below the peritoneal reflection. Urine and blood escape into the space of Retzius, spread upward toward the umbilicus, and laterally to the ischial spines. If the rupture is on the anterior wall, extravasation about the vesical neck and between the bladder and rectum will occur. This causes chills, fever, tenderness, infection, sloughing, and gangrene

- 4 Blood at the urethral meatus
- 5 Ecchymosis, contusions, and hematoma of lower abdomen, pubes, genitalia, and thighs.

Diagnosis is made on

- 1 History
- 2 Shock
- 3. Local pains, tenderness and swelling.
- 4 Inability to void.
- 5 Blood in urine
- 6 The passage of a catheter with the injection of a measured amount of fluid remeasured on its return is not a reliable test
- 7. Evidence of extravasation of dye in a cystogram.
- 8 Cystoscopic examination, which may reveal the tear.

Treatment.—

- 1 Combat the shock and hemorrhage which is most important

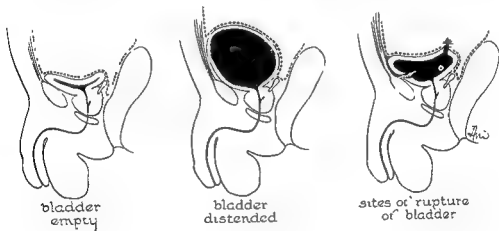


Fig 248 —Diagram showing varying anatomical relations of bladder when empty, distended, and ruptured

Symptoms of Rupture of the Bladder.—Early symptoms are due to injuries. Late symptoms are due to extravasation of urine and infection

Signs of Rupture are—

- 1. Shock
- 2. Helplessness, patient is unable to walk or walks with difficulty
- 3. Partial or complete inability to void

2 Explore the bladder by suprapubic cystotomy and provide drainage. This should be done even in doubtful cases

3 If the tear is intraperitoneal, open the peritoneal cavity, mop up any free fluid, and drain the peritoneum separately from the bladder.

4 If the tear is extraperitoneal, drain the bladder, and all the extravasical areas of extravasation

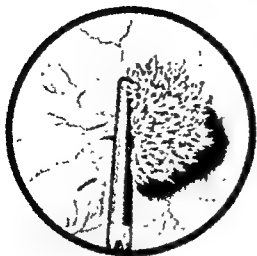


Plate XXXII.—Bladder Papilloma Close to Left Ureteral Orifice.

(Courtesy Victor F. Marshall: *The Diagnosis of Genito-Urinary Neoplasms*, American Cancer Society, Inc. From Pullen's *Medical Diagnosis*, W. B. Saunders Company)



Plate XXXIII.—Carcinoma of Urinary Bladder.

(Courtesy Victor F. Marshall: *The Diagnosis of Genito-Urinary Neoplasms*, American Cancer Society, Inc.)

They may be found anywhere in the urinary bladder. The majority are situated on the trigone or at the junction of the trigone and bladder. They frequently occur at the ureteral orifices. They are less common on the lateral walls.

Pathology.—*Gross*—The papilloma appears as a pedunculated villous or papillary growth arising from the mucous membrane. It may be sessile, but this type is less frequent. Some papillomas have a wartlike surface, while others are almost smooth. The size of the growths varies considerably; some are small and may remain so over long periods. Both the pedunculated and the more malignant sessile tumors may attain considerable size.

Microscopic—A typical papilloma shows multiple connective-tissue stalks containing thin-walled blood vessels, thus the tendency to spontaneous bleeding. Glandular or cystic formations may be present, or an inflammatory process may cause a leukocytic invasion of the central stalk, but these should not be mistaken for a malignancy.

Histologically there is no difference between the single and multiple forms. Ninety per cent are papillary in character. Many are benign, and may remain so for years. The marked tendency to malignancy of the so-called benign papilloma, and the ill-defined demarcation that exists between it and malignant growths have led many pathologists to regard all vesical papillomas as potentially malignant, even if initially they can be proved to be benign.

PAPILLARY CARCINOMA

Papillary carcinoma is a papilloma with malignant infiltration at the base and in the underlying bladder wall. It is the most common type of vesical carcinoma. The American Registry of Pathology classifies all papillomas as papillary carcinomas.

SQUAMOUS CELL CARCINOMA

(FLAT INFILTRATING CARCINOMA)

Squamous cell carcinoma is less common than papillary carcinoma. It is a flat sessile

growth with a cornifying epithelium which projects only slightly into the vesical cavity but invades deeply and spreads laterally. It is deeply and rapidly infiltrating at the outset. It is the most malignant of the vesical neoplasms and is radioresistant.

Pathology.—*Gross*—This carcinoma appears as a shallow red malignant ulcer surrounded by a hard everted border.

Microscopic.—Relatively thick connective tissue is seen between the cell nests, as distinguished from the papillary type of carcinoma. The cells progress along the surface of the bladder and at the same time invade the deeper layers of the wall.

Leukoplakia is commonly regarded as a precursor of squamous cell carcinoma.

ADENOCARCINOMA AND ADENOMA

Adenocarcinoma, primary in the bladder, is a rare, highly malignant neoplasm closely resembling adenocarcinoma of the prostate gland. It arises chiefly from the paraprostatic glands that are sometimes present in the wall of the male trigone, and in remnants of the urachus in the apex of the bladder. It may occur as a flat growth, showing extensive infiltration of the bladder wall, or it may have a thick pedicle and project into the bladder as a pedunculated tumor. It ulcerates early.

INTERSTITIAL CARCINOMA

An interstitial carcinoma growing in the wall of the bladder may not produce any symptoms except a decreasing bladder capacity. The bladder is rigid and will not stretch under anesthesia when one tries to introduce fluid forcibly. The growth is usually located in the vicinity of the bladder neck or at the apex. The structure varies greatly. They are usually small in size and sessile, though some are pedunculated.

MESOBLASTIC TUMORS

Benign—may be fibroma, myoma, myxoma, angioma, hemangioma or neurofibroma.

Sarcoma—is rare and fast growing

METASTASES

Metastases of bladder tumors occur in 10 to 40% of all cases. Metastasis may be

1. By direct local extension to the prostate, pelvic tissues, to the kidney via the ureter, to the penis or vagina.

2. By the lymphatic or blood stream to the long bones, liver, lungs, and regional lymph nodes. Recurrence after removal is fairly common.

Symptoms.—Painless hematuria is present in 80% of the patients. Frequency, dysuria, pyuria, and interruption of the urinary stream are other predominant symptoms.

Diagnosis is made on

1. History.

2. Physical examination.

3. Examination of urine for malignant cells with special stains.

4. Cystoscopy—the tumor is viewed directly.

5. Cystogram—filling defect may be seen

6 Biopsy.

Treatment.—

1. Fulguration or coagulation of the tumor either transurethrally or suprapubically, depending upon the size.

2. Local resection of the tumor-bearing area with 1 cm of healthy-appearing tissue around it

3 Local resection plus the implantation of radon seeds.

4 Radiation both externally and by opening the bladder and inserting an x-ray tube has been used, but produces a severe cystitis without appreciably affecting the tumor.

5 Total cystectomy with transplantation of the ureters to either the bowel or skin

6 Permanent suprapubic cystotomy.

Metaplasia of the Bladder

The epithelial cells of the urinary tract are particularly prone to proliferation and metaplasia. After minimal injuries such as an infection in the bladder or a calculus, epithelial changes may occur and result in projection of buds from the inferior layer of epithelium of the bladder into the sub-

mucosa. These are known as *Brunn's epithelial nests* which may become vacuolated, forming cysts. This is known as *cystitis cystica*. (Ureteritis cystica, or pyelitis cystica represents the same process going on in the ureter or the pelvis of the kidney.) Either of these conditions may be a precursor of leukoplakia and in turn of *squamous cell carcinoma*.

In other "Brunn nests," changes may occur that result in gland formation. The epithelial cells lining these glands are capable of secreting mucus. This process is known as *cystitis glandularis*, and may undergo a metamorphosis into a mucus-secreting *adenocarcinoma*.

Ulceration of the Bladder

May be due to:

1. Interstitial cystitis (submucous fibrosis).

2. Infections other than tuberculosis.

3. Carcinoma.

4. Radiation.

1. *Interstitial Cystitis*.—Synonyms: Hunter's ulcer, submucous fibrosis, elusive ulcer. This is characterized by single or multiple minute lesions of the bladder wall. Ulcers are not always present. The etiology of this disease is entirely unknown. It is much more common in females. The lesions are frequently located on the dome of the bladder. Pathologically the mucosa is flattened and there is submucous fibrosis. The clinical picture is variable, but usually there is pronounced frequency and suprapubic pain. Microscopically the urine is normal except for a few scattered red blood cells in it.

The treatment of this condition is often extremely difficult, and has led to a wide variety of methods:

(i) Dilatation of the bladder under spinal anesthetic.

(ii) Instillation of Novocain anesthetic into the bladder followed by silver nitrate.

(iii) Fulguration of the ulcers.

(iv) Resection of the bladder, removing the ulcerated areas.

2. *Infectious Ulceration*.—Ulceration associated with pyogenic infection of the blad-

3. Foreign bodies in the bladder.
4. Neurogenic bladder.

The general etiology of calculi has been discussed on page 559. In the obstructed bladder with its concomitant residual urine and associated infection, the *Bacillus proteus* is frequently the offending organism. A foreign body in the bladder such as a surgical sponge left after an operation on the bladder, or a broken tip of a filiform dilator is certain to produce a calculus. Many patients with a neurogenic or cord bladder following a spinal cord injury have some residual urine, which even though small in amount becomes heavily infected, thick and foul, and frequently causes stone formation. This can usually be prevented by the passage of a catheter and bladder lavage, once a week.

2. Phosphatic: calcium phosphate, magnesium phosphate, ammonium phosphate. The stone is oval or round, white in color, and may be friable.

3. Calcium oxalate. The stone is round, brown to black in color, has a mulberry surface, and is extremely hard.

Complications of Vesical Calculi.—

1. Obstruction of bladder neck.
2. Infection
3. Ulceration of bladder.
4. Impaction of the stone in the bladder neck, diverticulum or urethra.

Symptoms.—

1. Pain radiating to the glans penis, present on voiding and on exercise.
2. Frequency and urgency.
3. Hematuria.

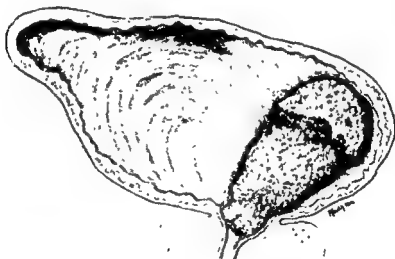


Fig 251 —Calculus in bladder, protruding into bladder neck

The chemical composition of a vesical calculus is generally mixed. Different compositions produce layers so that the stone presents a lamellated appearance in cross section

Composition of Vesical Calculi.—

1. Uric acid, urates.—The stone is flat or ovoid; yellow to dark red in color; friable, and may be either rough or smooth

4. Interruption of the urinary stream.
5. Retention of urine.

Diagnosis is made on:

1. History.
2. Urinalysis, examining especially for red blood cells
3. X-ray evidence of calculus in the bladder. About 95% of bladder calculi contain calcium and are visible radiologically.

der may occur, but is uncommon. Ulcers may be single or multiple, and disappear when the infection is eliminated. Ulceration in tuberculous cystitis is relatively common. See section on Genitourinary Tuberculosis.

3 Carcinoma.—The ulceration associated with carcinoma of the bladder is readily recognized. It is slow growing, clear cut, and appears punched out. Any ulcer of the bladder that becomes chronic should be suspected of malignancy.

4 Radiation Ulcer.—A radiation ulcer of the bladder may follow deep x-ray therapy to the pelvic region, or follow the implantation of radium into the cervix. The actual ulceration may not commence until months up to 12 years after the exposure. The ulcer is indolent, slow growing, and may progressively increase in size. The underlying pathological process is an endarteritis, with obliteration of the vessels. The treatment is symptomatic. Instillations of oil into the bladder give some relief of symptoms. Cerutin appears to have a beneficial effect on cases recognized early, before actual ulceration has occurred.

Diverticulum of the Bladder

A diverticulum or saccular ballooning out of the bladder wall is a relatively common urological finding. It may vary in degree from multiple shallow sacculations involving most of the bladder wall to single or multiple sacs connected to the bladder by a narrow opening. They may reach a considerable size. A diverticulum with a narrow neck drains poorly and almost always contains stagnant infected urine. The causes of diverticulum may be:

- 1 Congenital weakness of the bladder wall.

- 2 Obstruction to the urinary outflow (prostatic obstruction, urethral valves, etc.).

It is probably more nearly correct to say that a diverticulum is always due to obstruction of the outflow. A diverticulum may be complicated by the presence in it of a calculus or a tumor.

Symptoms.—The diverticulum itself rarely produces symptoms. It is usually a coincidental finding at the time of investigating a patient for symptoms of urinary obstruction. Pyuria is a common finding.



Fig. 250—X-ray showing diverticulum of bladder.

Treatment consists of

- 1 Removing the diverticulum or the mucous membrane lining it.

- 2 Removing the cause of the obstruction (prostate, vesical neck contracture, valves in the urethra, etc.).

- 3 The treatment of a calculus or a tumor in a diverticulum is to remove the diverticulum.

Vesical Calculus

Calculi in the urinary bladder may be single or multiple, they vary from the size of a garden pea to that of a full-term fetal head. They vary in shape from round to jackstones, and may be smooth or irregular. They are most commonly due to or associated with:

- 1 Infection.

- 2 Obstruction of the bladder neck, usually associated with infection.

guinea pigs by insemination with sperm from isolated epididymies.

Effects of Endocrine Therapy on Prostate Gland.—There is no verified clinical or experimental evidence that endocrine therapy has any effect on either the normal or hypertrophied adult prostate gland of man.

There is no positive clinical or experimental evidence that administered androgen or estrogen causes carcinoma in the prostate.

When estrogen is administered to patients with carcinoma of the prostate, there appears to be retardation of the growth, which may decrease in size.

Conditions of the Prostate Gland

ANOMALIES

Absence of the prostate gland or congenital cyst rarely occurs. A congenital cyst may be removed surgically.

PROSTATIC INJURIES

These may be due to

(i) External injury, e.g., falling astride a picket fence. Rupture of the urethra is a frequent complication, and is the usual cause of a stricture located in the posterior urethra.

(ii) Internal injury caused by the passage of instruments. A false passage may be made when passing a sound.

(iii) Operative injury, which is very rare, but may occur during operations on the perineum.

PROSTATIC CALCULI

Calculi form in the prostate gland only when an infection has been present for a prolonged period. The calculi are usually multiple, and vary in size from that of grape seeds to the size of marbles, completely replacing the glandular structure so that only the capsule is left. There may be hundreds of smaller calculi, or only a few large ones.

They may not cause any symptoms, or may cause typical symptoms of prostatic obstruction. They can frequently be palpated rectally, and visualized on x-ray.

Treatment.—If the calculi are small, they are removed by doing a transurethral prostatic resection. If they are large and fill the entire prostatic capsule, they should be removed surgically either by the suprapubic, retropubic, or perineal route.

PROSTATISM (SYNONYM: BENIGN

PROSTATIC HYPERTROPHY)

Definition.—Prostatism is a term used to describe a condition in which urinary symptoms develop as the result of obstruction at the bladder neck due to either an increase or decrease in the size of the prostate gland. It is caused by hyperplasia of the gland (glandular), sclerosis of the gland (median bar), or by invasion of the gland by new growth (carcinoma).

Age Incidence.—Prostatic obstruction occurs with increasing frequency in each decade, from the age of 40 onward. The incidence is 40 to 49 (9%); 50 to 59 (20%); 60 to 69 (35%); 70 to 79 (43%).

Theories of Origin.—The so-called prostatic hypertrophy is neither prostatic nor hypertrophy. It is hyperplasia. It consists of tissue which arises from the epithelium and the ducts surrounding the verumontanum on the floor of the urethra. The hyperplasia consists of nodules made up of fibromuscular and glandular tissue. The former arises from an analogue of the Müllerian duct system and the latter from the prostatic duct epithelium.

Chronic infection or vascular changes in the prostate gland and hormonal imbalance have also been thought to be responsible for the gradual increase in the size of the gland. Castration, or the administration of estrogenic substances, does not result in reduction of the hyperplastic process.

Symptoms of Prostatic Obstruction.—The first symptom of prostatic obstruction is the gradual onset of difficulty in starting the urinary stream, followed by increased frequency, nocturia, hesitancy of the urinary stream, urgency, and in many cases hema-

4 Cystoscopic examination with direct visualization of the calculus.

Treatment.—

1. Crushing of the stone instrumentally through the urethra if it is not too large (litholapaxy).

2. Removing the stone by opening the bladder suprapubically (cystolithotomy).

3. Removal of the calculus by perineal section.

Foreign Bodies in the Bladder

A wide variety of foreign bodies may be found in the urinary bladder. The com-

monest are surgical sponges, broken off tips of filiform dilators, and the wide variety of foreign bodies introduced by sexual perverts. Such objects may be toothpicks, lead pencils, chewing gum, hairpins, etc.

The presence of a foreign body in the bladder produces an intense acute inflammation with all the symptoms of an acute cystitis previously described. The foreign body can frequently be removed by instrumentation through the urethra. If this is not possible, suprapubic cystotomy is done, and the foreign body removed.

Extravasation of Urine (see page 574)

THE PROSTATE GLAND

Anatomy.—The prostate gland is a muscloglandular organ, which surrounds the neck of the bladder and the first portion of the urethra in the male. It is situated below the pubic symphysis and behind the inferior fascia of the triangular ligament. Its posterior surface rests on the rectum. The urethra traverses its length. The seminal vesicles which are situated on the posterior surface empty into the ejaculatory duct on each side in the prostatic urethra. The prostatic ducts also open into the prostatic urethra.

The prostate gland is made up of three lobes, two lateral and a middle, which give the gland a horse-collar shape. It normally weighs about 20 grams. Attached to the prostate are the puboprostatic ligament, and the inferior fascia of the urogenital diaphragm (triangular ligament). It is surrounded by a firm fibrous capsule.

The blood supply of the prostate is derived from the internal pudendal, vesical, and middle hemorrhoidal arteries.

The prostatic capsule is surrounded by a rich plexus of veins which terminate in the internal iliac vein.

Embryology.—At about the 55 mm. stage a series of buds arranged in 5 groups, arising from all sides of the urethra and from the

upper pelvic portion of the definitive urogenital sinus, grows into the dense surrounding mesenchyme which later differentiates into the muscular and connective tissues of the gland. The posterior lobe rudiment develops a separate capsule.

Normal Physiology of the Prostate

Prostatic Secretion.—The daily secretion averages 0.5 to 2.0 c.c. This is discharged through the ducts into the urine. Prostatic secretion contains on chemical analysis organic and inorganic substances, sodium, potassium, calcium, protein, glucose, ascorbic acid, citric acid, acid phosphatase, alkaline phosphatase, and cholesterol.

Acid phosphatase is present in very small amounts before puberty, but increases greatly during the sexual life. In carcinoma of the prostate, acid phosphatase appears in large amounts in the blood when the growth extends beyond the capsule of the gland.

The prostate does not have any internal secretion.

There is a large amount of lipid fluid in the prostatic secretion in the form of corpuscles which may be mistaken for pus cells.

Prostatic fluid is alkaline. It apparently contains no secretion which is essential to fertilization. Young produced pregnancy in

prostatic tissue is 500 to 2,500 units of activity per gram fresh tissue as compared with less than 5 units of activity at pH 4.9 for kidney, liver, duodenal mucosa and bones. The enzyme normally is excreted in the prostatic component of the ejaculate. Its exact function is unknown. In prostatic carcinoma, when the capsule of the gland ceases to be intact, and the growth infiltrates into the surrounding soft tissues or metastasizes to bone, the "acid" phosphatase which is normally below 3 units per 100 c.c. of serum rises. Values above 6 units per 100 c.c. are pathognomonic of metastasizing prostatic carcinoma.

When the secretory stimulus of the androgens is removed by castration, there is a rapid fall in the acid serum phosphatase by 45% within 48 hours, 73% within 2 weeks, followed by a transient gradual rise, and finally, a prolonged decline until after 2 or 3 months, equilibrium is reached. In those patients who do particularly well clinically, the acid serum phosphatase remains at these new lower levels. A persistently high postoperative acid phosphatase after castration is due to stimulation by androgens from extragonadal sources (adrenals).

The Significance of the Alkaline Serum Phosphatase in Prostatic Carcinoma.—There is no correlation between the acid serum phosphatase and alkaline serum phosphatase in patients with metastasizing prostatic carcinoma. The amount of alkaline phosphatase in the blood is an indication of the activity of the bone defense. After castration, the early changes are not consistent, but after a latent period of 2 to 3 weeks there is a rise of the alkaline phosphatase attributable to the osteoblastic activity in the healing of skeletal metastases. As the extra bone formation finally decreases, the serum alkaline phosphatase level declines to normal.

Symptoms of Carcinoma of the Prostate.—

1. Prostatism—causing symptoms of obstruction

2. Pain—due either to direct extension in the pelvis and involvement of nerve sheaths, or to metastases in the spine or pelvic bones.

Treatment.—

1. Radical perineal prostatectomy in cases recognized early. This is the only method of obtaining a cure. The prostate, seminal vesicles, and bladder neck are removed en masse.

2. Surgical castration.

3. Medical castration—by giving Stilbestrol 5 mg. per day orally for 2 weeks, then 5 mg. twice a week thereafter.

Some surgeons prefer to bombard the prostate with large doses of estrogen, thereby causing the gland to shrink, and to follow this by a radical perineal prostatectomy. It is claimed that an inoperable carcinoma is thereby converted into an operable one.

In patients suffering excruciating pain from metastases, spectacular and complete relief of pain is obtained, in most cases within 72 hours, by surgical castration.

Chronic Prostatitis and Nonspecific Urethritis

Nonspecific urethritis and prostatitis are frequently erroneously regarded as being a chronic form of gonorrheal urethritis. Either or both may exist without a previous attack of gonorrhea. Chronic nonspecific urethritis alone is rare, whereas chronic prostatitis alone is common. Fifty per cent of cases with chronic prostatitis or urethritis give a previous history of gonorrhea; 36% have had what was diagnosed as a nonspecific urethritis; and 14% have never had any previous symptoms, the discovery of a large number of pus cells in the prostatic fluid being a coincidental finding. In this latter group the infection is comparable to an upper respiratory infection except that the infection settles in the prostate gland which because of its dependent position in the body with consequent poor drainage,

turia Incomplete retention develops (i.e., residual urine) and eventually a complete retention with inability to void. Some cases show incontinence due to stretching of the internal sphincter by the increased size of the prostate. Sexual debility is a common complaint. There may be pain either in the suprapubic region or the lower lumbar region posteriorly.

Diagnosis of Prostatic Obstruction.—A diagnosis of prostatic obstruction is made on the history of gradually increasing urinary difficulty and the symptoms listed above. On rectal examination an enlarged prostate is palpated. The patient is catheterized immediately after voiding to determine the amount of residual urine present. On cystoscopic examination the enlarged prostate is visualized at the bladder neck. A cystogram will also frequently show an intravesical protrusion of the enlarged prostate.

Treatment.—There are four accepted surgical methods of treating enlargement of the prostate gland.

1. *Transurethral Resection*—In this operation successive pieces of tissue are removed from around the bladder neck and the prostate itself by means of a resectoscope. The gland is not as completely removed as in the other types of operation.

2. *Suprapubic Prostatectomy*—This may be carried out in either one or two stages. A skin incision is made in the suprapubic region of the lower abdomen, the bladder opened, and a drainage tube inserted into the bladder. The prostate is removed at the time of opening the bladder or at a subsequent operation 2 weeks or more later. If the patient is debilitated or uremic, removal of the prostate is deferred until the patient improves.

3. *Retropubic Prostatectomy*—The prostate gland is removed through a skin incision made in the lower abdomen. The entire procedure is done outside the bladder through the prostatic capsule, without actually opening the bladder.

4. *Perineal Prostatectomy*—The prostate gland is removed through an incision in the perineum above the anus which exposes the gland from the outside, without entering the bladder.

Carcinoma of the Prostate

Incidence.—Carcinoma of the prostate gland affects 5% of all men who reach the age of 60 years. One of every five prostate glands removed for benign hyperplasia shows carcinoma histologically.

Carcinoma usually begins in the posterior lobe, but may be found anywhere in the gland.

Diagnosis of Carcinoma of Prostate.—This is based on:

1 Rectal examination. The gland is usually irregular in shape and stony hard in consistency. The hardness may be limited to a small area of the gland, or involve the entire gland, even extending out laterally into the ligaments and upward into the seminal vesicles. Such a gland is referred to as "fixed" or "frozen."

2. Examination of the prostatic fluid by special stains for carcinoma cells is unreliable even in proved cases of carcinoma.

3 Biopsy of the prostate gland establishes absolute proof of carcinoma, but is occasionally misleading, since the biopsy may fail to show carcinoma which is present elsewhere in the gland.

4 X-ray evidence of metastases in the bones or chest.

5 Elevated acid serum phosphatase.

The Significance of the Acid Serum Phosphatase in Prostatic Carcinoma.—A phosphatase is an enzyme which splits organic phosphorus compounds to give free phosphate ions. Phosphatases differ in the pH at which they show their maximum activity, and accordingly are known as *acid* or *alkaline* phosphatases.

Normal human prostatic tissue is the only tissue in the body that is rich in *acid phosphatase*. The concentration in adult human

Prostatic massage is considered to be an essential part of the treatment. When this is deleted from the program of treatment, the percentage of cases "apparently cured" drops from 75% to 10%.

Treatment may require weeks or even months to complete. Recurrence is unfortunately common, and suggests that the disease was not entirely eradicated by treatment, despite clinical and laboratory tests to the contrary.

Prostatic Abscess

Etiology.—Prostatic abscess may be single or multiple, and follows:

1. Acute gonorrheal urethritis (uncommon now).
2. Any nonspecific prostatitis
3. Traumatic instrumentation.
4. Urethral stricture.

5. Metastatic, as a complication of pyemia, typhoid fever, influenza, carbuncles or boils.

Symptoms.—The symptoms are pain in the perineum, fever, chills, and increasing difficulty in voiding which may progress to acute retention.

Prognosis.—The prostatic abscess may rupture into the urethra, rectum, perineum, or bladder.

Treatment.—

1. Incision and drainage of the abscess through the perineum. A perineal urethrotomy may be done at the same time and an indwelling catheter inserted.

2. Chemotherapy—usually one of the antibiotics, the type depending upon the bacteriology of the infection.

Tuberculosis of the Prostate

See page 567.

THE PENIS AND URETHRA

Conditions of the Penis

1. Congenital anomalies (double phallus, hypospadias, epispadias).
2. Phimosis, paraphimosis, balanitis.
3. Venereal warts (condylomata acuminata, condylomata lata)
4. Chancre, chancroid, lymphogranuloma venereum, granuloma inguinale
5. Plastic induration (Peyronie's disease)
- Priapism
7. Tumors.

Congenital Anomalies

Double phallus is a rare anomaly.

Hypospadias is a congenital defect of the anterior urethra. The urethra is incompletely formed so that it opens on the under surface of the penis, in the scrotum or perineum. This is a relatively common anomaly. Hypospadias is classified as first, second or third degree, depending upon the site of the urethral opening. The first degree open-

ing is just behind the normal site of the external urinary meatus. In hypospadias a hood or cap of foreskin overhangs the glans penis. This should be preserved for future use in plastic procedures on the penis. Associated with the hypospadias there is a marked chordee or downward curvature of the penis, caused by replacement of the corpus cavernosum urethrae by fibrous tissue. Correction of the hypospadias is achieved by a series of plastic operations. The first stage consists of removing the scar tissue causing the chordee and straightening the penis. At the second operation a new urethra is formed by using full or split thickness skin grafts, or by means of a tube pedicle graft. The urethra is buried, covered, and finally connected to the normal urethra. Before such operations are undertaken, the urinary stream is diverted by suprapubic cystotomy. The end results are usually satisfactory.

and complex histological architecture, affords an ideal environment to harbor infection.

Symptoms.—The patients' symptoms are urethral discharge, frequency, burning, terminal hematuria, vague perineal discomfort and backache. In the group of cases in which there is a previous history of gonorrheal or nonspecific urethritis, urethral discharge is usually present, and is the main symptom in over 70% of cases. In those cases in which there is no previous history of urethritis, a urethral discharge occurs in approximately 21%. Pyuria without symptoms is a common finding.

Origin of Condition.—The majority of these patients give a history of alcoholism and sexual excess immediately preceding the onset or recurrence of symptoms. This appears to be a factor in lowering the resistance of the urethra and prostate by

(i) Favoring subsequent invasion by organisms from the host which are normally nonpathogenic, via the lymphatics or possibly the blood stream.

(ii) Exacerbation of a previous prostatitis in which there is still some residual infection present.

(iii) Organisms from a carrier at the time of intercourse. The organisms may not be pathogenic to the carrier.

(iv) Virus.

Bacteriology.—The urethral discharge when cultured shows staphylococci in 55% of cases, diphtheroids 10%, mixed staphylococci, streptococci, and micrococci 32%, trichomonas vaginalis 2%, and gonococci 1%. Protozoa may also be found in the urethral discharge.

Diagnosis.—A diagnosis is made on

1. The history and symptoms
2. The presence of urethral discharge.
3. The three-glass urinalysis
4. Examination of the prostatic fluid

The symptoms of urethral discharge, frequency, burning, terminal hematuria, and vague perineal discomfort, are almost

pathognomonic of the condition. Palpation of the prostate is often misleading. In some cases the gland is firm and fibrosed; in others it is soft and normal to palpation. Examination of the prostatic fluid obtained by massage is essential to establish a diagnosis.

Criteria of Cure.—

1. The patient must be entirely free of symptoms and there must be no urethral discharge.

2. The three-glass urinalysis is normal.

3. The prostatic fluid on culture is negative for gonococci.

4. The number of pus cells in the prostatic fluid is reduced to below 5 per high power field.

5. Re-examination in three months' time confirms these findings.

Treatment.—

1. Prostatic massage—twice weekly—until apparently cured.

2. Dilatation of urethra—stricture of the urethra even though not severe, and a small external urinary meatus, are the two commonest causes of failure to eradicate the infection. A normal urethra should accommodate a French No. 26 sound without difficulty.

3. Urethral irrigations are frequently helpful, particularly if there is much urethral discharge. Any one of several solutions may be used.

(i) Protargol— $\frac{1}{4}$ of 1%

(ii) Potassium permanganate—1 in 12,000 solution

(iii) Silver nitrate—1 in 10,000 solution.

4. Chemotherapy

(i) Penicillin—300,000 units of penicillin G intramuscularly daily for 14 days.

(ii) Streptomycin 1 gram daily for 14 days

(iii) Aureomycin—according to weight

(iv) Chloromycetin—according to weight

(v) Terramycin.

(vi) Atabrine, gr. iss t i d.

(vii) Arsenic

glands are enlarged, firm, and discrete (shotty). The treatment is antisiphilic therapy.

Chancroid (Soft Chancre).—Chancroid is a venereal ulcer usually situated on the external genitalia and caused by the Ducrey-Unna bacillus. Uncleanliness and dirty habits favor its development. The ulcers are irregular with a grey base and undermined edges. Suppurative inguinal adenitis may occur and is called a bubo.

Diagnosis is made on the appearance of the ulcer, the clinical course of the disease, the skin test (commercial antigen injected intradermally), and the presence of the Ducrey bacillus. Syphilis must be excluded.

Treatment consists of local cleanliness and sulfonamide therapy. Cauterization of the ulcers with nitric acid has been recommended.

Lymphogranuloma Venereum is a specific disease caused by a filtrable virus. A primary sore appears 2 to 7 days after exposure usually on the corona in the male and on the posterior vaginal wall or posterior cervical wall in the female. The initial lesion is vesicular or nodular. Soon there is swelling in the inguinal nodes. The overlying skin breaks down and sinuses form. Diagnosis is made upon the clinical appearance and the Frei cutaneous test.

Antimony is specific for this disease. Aureomycin is also effective.

Granuloma Inguinale is a lesion which begins as a papule in the groin, perineum, vulva or prepuce. It soon ulcerates and invades the surrounding tissues. It does not show any tendency to heal. It is caused by a specific organism called the Donovan body (*Bacillus mucosus capsulatus*). Secondary infection and erosion cause marked deformity of the organs. A diagnosis is made by the presence of Donovan bodies. Specific treatment is the intravenous administration of antimony and potassium tartrate. Fuadin is also effective.

Plastic Induration of the Penis (Peyronie's Disease)

The induration is due to fibrous infiltration of the penis which begins in the septum between the corpora cavernosa. It may extend into any part of the penis, and finally reaches Buck's fascia and the tunica albuginea where it can be felt as hard indurated plaques. These plaques and fibrosis cause deformity of the penis during erection and marked pain. The etiology is unknown.

Various forms of treatment have been recommended including, radiation therapy, excision, plastic procedures, etc. The results are moderately satisfactory.

Priapism

Priapism is prolonged involuntary erection of the penis without sexual desire. This is an uncommon condition, and difficult to treat, as it does not respond to medication. It may occur at any age, but is more common in young adults. There is usually some serious underlying disease, such as leukemia, syphilis, neurological disorders, or malignancies. Treatment consists of treating the underlying cause if one can be discovered. Incision of one or both corpora with expression of blood clots, which form after a prolonged period, may be required.

Prognosis.—*Impotence usually follows.*

Tumors of the Penis

Benign.—

- (i) Condylomata acuminata (venereal warts). See previous description.
- (ii) Condylomata lata (syphilis). See previous description.
- (iii) Angioma
- (iv) Balanitis xerotica obliterans is a weeping dermatitis of the glans penis, regarded as premalignant.

Malignant.—

- (i) Carcinoma (squamous cell carcinoma).

Epispadias is a rare anomaly. The urethra is situated above the corpora cavernosa and lies open on the dorsal surface of the penis. The roof of the urethra is lacking throughout part or all of its length. This condition is usually associated with extrophy of the bladder which may be partial or complete. Surgical correction of the deformity with construction of a urethra and closure of the bladder can be carried out, although incontinence is likely to persist. An alternative treatment is to transplant both ureters into the bowel and excise the bladder.

Phimosis

Phimosis is a condition of the foreskin which prevents its retraction. The prepuce is long and redundant. The acquired form is seen in adults and is due to irritation which may be inflammatory, traumatic, or consequent to incontinence associated with prostatic obstruction or overflow from the bladder.

In the acute stage irrigations under the foreskin with potassium permanganate or boracic acid solution hasten recovery. If the foreskin is sectioned along the dorsum of the glans penis, the healing process is further accelerated. When the acute inflammation subsides, circumcision is indicated to prevent recurrence.

Paraphimosis

Paraphimosis is strangulation of the foreskin by the circular constriction of the retracted prepuce which can no longer be reduced. It frequently accompanies acute inflammatory conditions, such as gonorrhea, chancre, and chancroid. It requires immediate relief either by digitally reducing the glans through the ring or by surgical division of the constricting band (dorsal slit). Gangrene results if the strangulation is not relieved. Paraphimosis also occurs in the aged, when the elasticity of the foreskin is lost.

Balanitis

Balanitis is a superficial inflammation of the glans penis. In most cases the under-surface of the prepuce is also affected producing a balanoposthitis. The glans penis becomes damp, red and itchy. An exudate forms. Treatment consists of local cleanliness. Circumcision during quiescence is desirable so that debris and moisture can no longer collect beneath the foreskin.

Venereal Warts

Condylomata Acuminata are single or multiple wartlike eruptions on the skin of the external genitalia. They are variable in shape and are most commonly situated around the corona (i.e., the sulcus proximal to the glans penis) and on the prepuce. They are not due to venereal disease but are most commonly due to excessive moisture. They are removed by treating them with aqueous podophyllin 25%, silver nitrate stick, or by electro-fulguration. Clinically the results are much more satisfactory if the redundant foreskin with attached warts is excised, and the remaining warts fulgurated.

Condylomata Lata, the warts of secondary syphilis, are moist broad warts that frequently fuse and cover an extensive area, even extending as far as the anus. Diagnosis is based on dark-field and serological examination. Treatment consists of local cleanliness and antiluetic therapy.

Chancre, Chancroid, Lymphogranuloma Venereum

Syphilis (Chancre).—Syphilis is the result of infection by the *Spirochaeta pallida*. In the primary stage, at the end of the incubation period, a chancre appears on the glans penis or shaft of the penis, at the corona, frenulum or inside the prepuce. The lesion is painless. It is commonly single, but may be multiple. The chancre has a hard, indurated, raised edge, like a button, with an intensely red ulcerated center. The inguinal



Plate XXXVIII.—Condylomata Acuminata (Venereal Warts).



Plate XXXIX.—Carcinoma of the Penis.

(Courtesy Victor F. Marshall: *The Diagnosis of Genito-Urinary Neoplasms*, American Cancer Society, Inc.)

- (ii) Sarcoma—very rare
- (iii) Multiple hemorrhagic sarcomas—very rare.

Carcinoma of the Penis

Carcinoma of the penis is a relatively common malignant tumor. It is thought that accumulated smegma and uncleanness are predisposing factors. It is practically never found in the Jewish race or Mohammedans, both of whom practice circumcision as a religious rite in the first week of life. Strangely enough, if circumcision is performed on a child of 5, it does not prevent him from developing carcinoma in adult life, suggesting that whatever the carcinogenic factor may be, it remains latent throughout most of the life's span. Equally interesting is the fact that carcinoma of the cervix is relatively uncommon in Jewish women. The exact etiology of carcinoma of the penis is unknown.

Site.—The majority of carcinomas of the penis arise on the glans, or in the coronal sulcus. The lesion may be papillary, cauliflower, or infiltrating in type. Any of these may ulcerate. Metastases to the inguinal nodes occur early in the disease. Secondary infection of the nodes results in suppuration and necrosis.

Diagnosis is made on the clinical appearance and biopsy. Early diagnosis is important.

Treatment.—

1. Partial amputation of penis
2. Complete amputation of penis with radical excision of the inguinal lymph nodes
3. Caustic excision of the tumor with implantation of radon seeds
4. Irradiation

CONDITIONS OF URETHRA

1. Urethritis (gonorrhea, nonspecific, Reiter's disease)
2. Stricture.

3. Periarethral phlegmon (periurethral abscess).

4. Tumors.

5. Rupture

Urethritis

Etiology.—

Gonorrheal Urethritis is an acute specific inflammatory process involving the anterior urethra and caused by the Neisserian organism (gram-negative diplococci). The inflammatory process is characterized by an exudation of phagocytes in an endeavor to limit the infection. The production of pus results in a profuse urethral discharge. The glands of Littre in the anterior urethra are involved; this explains why an inflammatory stricture is found only in the anterior urethra.

Earlier teaching stressed the importance of avoiding spread of the infection to the posterior urethra and to the prostate by urethral irrigations or instrumentation. With modern chemotherapy in which neither irrigations nor instrumentation are used, spread of the inflammatory process to the posterior urethra and the prostate appears to occur spontaneously in some cases within the first 24 hours.

Symptoms of an acute gonorrheal urethritis are a profuse urethral discharge, frequency, burning, and urgency.

Diagnosis is made on the history of exposure and the finding of gram-negative diplococci either intra- or extra-cellular, or both, in smears of urethral discharge.

Complications of Gonorrheal Urethritis.—

1. Epididymitis. Elevate the scrotum and apply heat.
2. Prostatitis—acute, may become chronic.
3. Prostatic abscess.
4. Urethral stricture.
5. Arthritis
6. Gonorrheal endocarditis—very rare.

Treatment—(in order of preference)

1. Penicillin—300,000 units daily for 2 days



Plate XXXVIII.—*Condylomata Acuminata* (Venereal Warts).



Plate XXXIX.—*Carcinoma of the Penis.*

(Courtesy Victor F. Marshall: *The Diagnosis of Genito-Urinary Neoplasms*, American Cancer Society, Inc.)

2. Streptomycin—1 Gm. daily for 2 days.
3. Sulfonamides—either tri-sulfa, or sulfadiazine 4 Gm. daily for 3 days, gradually decreasing.
4. Irrigations—Protargol $\frac{1}{4}$ of 1%; potassium permanganate 1 in 12,000 solution; silver nitrate 1 in 10,000 solution—Irrigations are no longer used in acute or newly acquired infections, but are of considerable value in the chronic or recurrent form.

Nonspecific Urethritis (See page 583).

Reiter's Disease.—Reiter's disease is characterized by the triad, (1) urethral discharge, (2) conjunctivitis, (3) polyarthritis.

Its cause is unknown. No specific organism has been recovered from the urethral discharge. Conjunctivitis may be severe, and progress to erosion. Treatment is not specific and must therefore be symptomatic and palliative. Recovery occurs after a period of months.

Stricture of the Urethra

Stricture of the anterior urethra is usually inflammatory, but may be due to tearing of the urethra by a blow. Stricture of the posterior urethra is always traumatic in origin.

Diagnosis.—

1. History of a previous acute inflammation or trauma.
2. History of a narrow urinary stream, or forking of the stream
3. Signs of infection (frequency, urgency, burning, associated with a narrow stream).
4. Palpation of the urethra—an indurated area can often be palpated.
5. Urethrogram—x-ray of the urethra taken at the time of injection of an opaque liquid or jelly. The narrowed area in the urethra is visualized.
6. Calibration of the urethra with a bougie-à-boule. One can determine the site, length, and size of the stricture with this instrument.

Treatment of Stricture.—

1. Dilatation of the urethra may be carried out with filiforms and followers (gum elastic dilators attached to a fine guide); Philip's bougies, metal sounds (Laforge's), or by a Kollmann dilator.

2. Internal urethrotomy—A specially designed urethrotomy knife is passed down the urethra to the site of the stricture and a longitudinal incision of the stricture is made.

3. Surgical excision of the stricture—with end-to-end anastomosis, or tube graft.

Instrumentation of Urethra

1. For urinary retention.
2. For stricture of urethra.

Any instrumentation of the urethra should be preceded and followed by a thorough irrigation of the urethra with a mild antiseptic. Urethral chills with high fever due to toxic absorption are practically never seen if this precaution is taken. Sterile technique should be maintained throughout. Adequate lubrication is essential. Petroleum jelly, liquid lubricant, or olive oil instilled into the urethra are all satisfactory.

TECHNIQUE OF INSTRUMENTATION

Catheterization of the urinary bladder may be required for acute retention due to prostatism or following surgical operations. There are several different types of catheters with which this procedure may be carried out:

- (i) Soft rubber catheter, either plain or Foley bag type.
- (ii) Soft rubber catheter with catheter stylet.
- (iii) Coudé catheter (one angle on distal end).
- (iv) Bicoudé catheter (two angles on distal portion).

In patients with postoperative retention of urine or retention due to prostatism, an attempt is first made to pass a soft rubber catheter. This may be either a plain catheter or one with a distendable balloon on the

end (Foley bag catheter) if it is desired to leave it in place as a retention catheter. The urethra is well irrigated, and the catheter lubricated (either with sterile liquid paraffin, glycerine, or olive oil or lubricating jelly). The catheter is then passed *gently*. This is successful in the majority of cases. Additional lubrication of the urethra by the injection of 5 to 10 c.c. of the sterile lubricant into the urethra prior to passing the catheter, often converts a difficult into an easy catheterization.

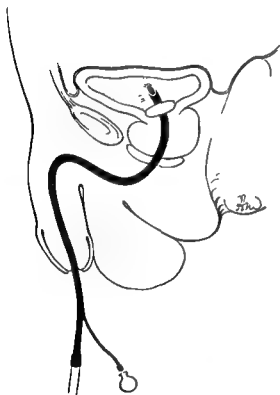


Fig 252—Foley bag catheter in the bladder

In cases of prostatism it may be impossible to pass a soft rubber catheter. A curved catheter stylet may be used to increase the rigidity of the catheter. The catheter with the stylet is guided into the bladder over the obstructing prostatic lobe.

If the above two maneuvers are not successful a *coudé* or *bicoudé* catheter is passed. These are gum elastic silk woven catheters with one bend about $\frac{1}{2}$ inch from the tip

(*coudé*) or two bends—one-half inch from the tip and the other three inches from the tip (*bicoudé*). They are most helpful in cases of prostatic obstruction.

For many years it has been taught that decompression of the bladder should be carried out very slowly and gradually over a 24-hour period, in order to avoid sudden massive hemorrhage. Sudden massive hemorrhage following rapid decompression is so rare that most urologists in the course of a lifetime do not see a case.

A *Bougie-à-boule* is an instrument about 10 inches long, with a narrow shaft, and an acorn-shaped enlargement at one end and an olive-shaped enlargement at the other end. The enlargements at the end are calibrated in size from French No. 14 to French No. 24. The instrument may be either gum elastic silk woven, or metal. It is used to explore the urethra to determine the presence of stricture. Its advantage over a sound is that tissue-paper-thin strictures can be palpated, whereas with a sound they would be broken down without being recognized. The largest size (F 24) is passed first. If an obstruction is encountered, the instrument is withdrawn and the next size passed, continuing thus down the scale until one is found that can be passed to the bladder. On withdrawing the bougie, the enlarged end is felt to grate and pass irregularly in the scar tissue of the stricture. It is thus possible to chart the site, length, and caliber of the stricture. Such information is valuable in subsequent treatment.

Metal Sounds.—The usual metal sound is an instrument that should be used with great delicacy and care. Under no circumstances should force be used. The urethra is irrigated and the instrument (F 12 or 14) lubricated. The point is passed through the meatus, the handle of the instrument being held over the patient's abdomen. As the deep urethra is approached the handle is rotated through an arc of approximately 120° downward, the curved proximal end of

the sound thus sliding under the symphysis into the bladder. The sound should be closely opposed to the roof of the urethra throughout its entire passage.

Kollmann's dilator is a metal instrument with expanding blades that are controlled by a screw arrangement at the head. It is passed like a sound. A scale also attached to the head of the instrument indicates the caliber obtained as the blades are opened. This dilator is seldom used.

Periurethral Abscess (Periurethral Phlegmon)

Periurethral abscess may occur at the site of any inflammatory stricture or when an indwelling urethral catheter is used for a prolonged period and causes erosion of the urethra.

Extravasated urine spreads into the scrotum, causing marked swelling of the scrotum and penis, and into the abdominal wall. It is limited in its spread by the attachment of Colles' fascia inferiorly to the lower border of the urogenital diaphragm, laterally to the rami of the ischium and pubis, superiorly it spreads within the abdominal wall deep to Scarpa's fascia.

The treatment of periurethral abscess and extravasation of urine consists of—

- 1 Diversion of the urinary stream by suprapubic cystostomy
- 2 Incision and drainage of the abscess.
- 3 Multiple incisions with through-and-through drainage in the area of extravasation

New Growths of the Urethra

New growths of the urethra, benign or malignant, are very rare, 192 cases of primary carcinoma of the male urethra have been reported

Symptoms and Signs—

1. Difficulty in micturition is the commonest symptom of growths in the anterior urethra.

2. Hematuria is the commonest symptom of growths in the posterior urethra.

3. The growth may be palpable.

The dysuria when associated with a periurethral mass and difficulty in dilating a previously easily dilated stricture are suggestive of new growth.

Diagnosis is made on

1. Symptoms.
2. Palpation of the urethra.
3. Endoscopic examination.
- 4 Biopsy.

Treatment.—

1. Amputation of the penis is the treatment of choice.

2. Extensive resection of the urethra.

3. Radium may be applied directly for a relatively small growth in the fossa navicularis.

4. Fulguration of small growth.

Prognosis.—Carcinoma of anterior urethra—50% of patients are living after 2 years. Carcinoma of posterior urethra—50% of patients are dead one year after operation, indicating the difficulty of complete removal of the growth if situated posteriorly

Urethral Caruncle is a growth resembling a thrombosed vein which appears in the female at the external urinary orifice. It is relatively common, benign, and treated by excision or fulguration

Rupture of the Urethra

Rupture of the urethra is caused by direct violence (such as falling astride a beam) or by indirect violence in crushing injuries of the pelvis. The site of the rupture is commonly in the membranous or prostatic urethra.

Signs of Rupture.—

1. Bleeding from the external urinary meatus.
2. Hematoma at the site of injury.
3. Inability to void.
- 4 Variable degrees of shock.

Treatment.—Depending upon the conditions present at the examination, one of several methods may be used to treat the ruptured urethra

1. If a soft rubber urethral catheter can be passed it should be left in place and taped in.

2. Immediate suprapubic cystotomy with repair of the urethra. The bladder is opened suprapubically and a sound is then passed through the bladder and internal sphincter into the urethra to the site of rupture. Another sound is passed through the external meatus into the urethra until it meets the former (male and female sounds). The sound passed from the bladder side is used to direct the other sound into the bladder. When this has been achieved, a catheter is attached to the sound passed through the penis and the sound and catheter are withdrawn in a retrograde manner. The catheter is left in situ as a splint for the urethra. A de Pezzer tube is inserted into the bladder and brought out through the suprapubic incision.

3. Immediate repair by exposing the site of rupture in the perineum, identification of the torn ends of the urethra, and end-to-end anastomosis of the ends over a rubber or Foley bag catheter.

4. Suprapubic cystotomy only, with repair of the urethra at a later date.

5. Perineal section and urethrotomy. A catheter is passed through the urethrotomy opening to the bladder to divert the urine. The urethra is repaired at a later date.

6. In the late cases, or in cases of old injury or those previously treated by suprapubic cystotomy, the scarred torn urethra is exposed in the perineum. The scar is excised, and a split thickness skin graft is built up over a tracheal cuff placed on a Foley bag indwelling catheter. The cuff with its attached skin graft is then positioned on the catheter to fill the gap caused by the excision of the scar. Two inches or more of the urethra can be replaced by this method.

All cases of rupture of the urethra will require periodic dilatation throughout the remainder of life.

SCROTUM AND SCROTAL CONTENTS

Congenital Anomalies

Anorchidism—congenital absence of both testes

Monorchidism—development of only one testicle.

Synorchidism—the fusion of both testicles intra-abdominally has only been discovered at operation or autopsy

Polyorchidism—supernumerary testicles

Cryptorchidism—congenital malposition of the testicle. The congenital anomalies with the exception of cryptorchidism are rare.

Surgical Conditions of the Scrotum

1. **Injuries.**—The scrotum may be subjected to a direct blow, lacerating wound or avulsion. A laceration is thoroughly cleaned with soap and water, debrided, and sutured

Avulsion of the entire skin of the scrotum may occur in industrial accidents where wheels and belts are employed. The treatment is

(a) immediate treatment of shock, followed by

(b) covering the scrotum with skin flaps

2. **Inflammation.**—Inflammation of the scrotal wall usually occurs secondary to general diseases such as diabetes, cardiorenal failure, etc. It may also occur as the result of a periurethral phlegmon, extravasation of urine, chemical or physical trauma (e.g., frost bite).

3. **Tumors.**—Tumors of the scrotum are very rare. The treatment is excision.

4. **Elephantiasis** in which there may be enormous swelling of the scrotum is due to

infestation by filariae which fill the lymphatic channels, causing obstruction. The disease is rare in temperate, but common in tropical, climates. The enlarged scrotum may reach the knees. Treatment consists of excision of the burdensome excess tissue.

Surgical Conditions of the Scrotal Contents

VESTIGIAL STRUCTURES IN THE SCROTUM

1. Appendix testis (i.e., hydatid of Morgagni) is pedunculated and attached to the front of the head of the epididymis, or lies in the groove between the testis and epididymis.

2. Appendix epididymis is a pedunculated hydatid. It is rare. It is attached to the front of the upper pole of the epididymis.

3. Paradidymis or organ of Giralde is a collection of small tubules found above the head of the epididymis and in the lower end of the spermatic cord in front of the vessels.

4. Vasa aberrantia or ductuli aberrantes, are coiled canals with blind ends in the epididymis. The inferior (that of Haller) is the larger and is located in the tail of the epididymis.

TESTICULAR TUNICS

1. Hydrocele.—A hydrocele is a sac of fluid within the scrotum, surrounding the testicle, the sac being formed by the tunica vaginalis. This is the commonest type and is called *idiopathic vaginal hydrocele*. In *congenital hydrocele*, the processus vaginalis communicates with the abdominal cavity. Elevation of the scrotum causes the fluid to return to the peritoneal cavity. In *infantile hydrocele* the tunica vaginalis extends up to the internal inguinal ring. The sac, however, has no connection with the peritoneal cavity. *Encysted hydrocele of the cord* is associated with the spermatic cord. The processus vaginalis is closed above and below, so that the sac moves with the cord when traction is applied to the testicle. A *secondary hydrocele* may occur as a complication of epididymo-orchitis, either acute or chronic.

Symptoms.—The patient notices a gradually increasing painless swelling in the scrotum.

Diagnosis is made on the presence of a scrotal mass and by transillumination. The spermatic cord and the external inguinal rings are normal to palpation and do not suggest an inflammatory process. In a hematocele, light is not transmitted. An epididymitis is tender to palpation.

Treatment.—

(1) **Aspiration** by means of a large caliber needle. This procedure must be repeated at intervals. It is not curative; the hydrocele recurs and the sac gets thicker.

(2) **Injection.** A needle is inserted into the sac and the fluid is withdrawn. Quinine urea hydrochloride at intervals of one week is injected into the sac on 2 or 3 occasions. Some claim permanent cure by this treatment.

(3) **Excision of the hydrocele.** The sac is opened, everted, and the excess wall is excised. The edges are sutured behind the epididymis.

(4) A scrotal support often provides comfort.

2. **Hematocele.**—This is a collection of blood in the tunica vaginalis. It usually results from an injury. A common cause is the aspiration of a hydrocele. Repeated hemorrhages may occur giving gradual enlargement. The testicle is flattened by pressure. Treatment consists of opening the sac, removing the clot, and excising the sac.

3. **Chylocele.**—Chylocele is the presence of chyle fluid (lymphatic fluid which contains cholesterol particles from the digestive tract) in the tunica vaginalis. It is rare, but may occur in cases of chronic lymphatic obstruction, e.g., filariasis.

4. **Hernia.**—A hernia containing intestine may be present in the scrotum as well as a hydrocele. It is one of the more important conditions to be differentiated from hydrocele. It does not transilluminate.

EPIDIDYMIS

1 **Injuries to the epididymis** are rare, but may follow a direct blow. Swelling and edema occur.

2 **Spermatocele and Cysts.**—Cysts of the epididymis, particularly the head, are much more common than spermatocele. They are usually multiple and filled with a clear fluid. A spermatocele is a unilocular retention cyst derived from some portion of the sperm-conducting system of the epididymis. It is therefore filled with cloudy fluid. Treatment is excision (of the cyst or spermatocele).

3. Inflammation.—

Gonorrhea—Acute gonorrheal epididymitis occurs as a complication of gonorrheal urethritis. The epididymis becomes enlarged, hard, and exquisitely tender. Pain is acute. Fever of 103° to 104° F. is often present. When epididymitis is acute, urethral discharge is scanty.

The *diagnosis* of epididymitis is made on

- (i) History of exposure
- (ii) An enlarged tender mass found on physical examination.
- (iii) High fever.
- (iv) The finding of Neisserian organisms in the urethral discharge

Differential Diagnosis—Epididymitis must be differentiated from torsion of the testicle. Prehn has described a useful sign in this connection, namely, that elevation of the scrotum gives relief of pain in epididymitis, but increases the pain in torsion of the testicle (Prehn's sign).

Treatment —

1. Specific—penicillin or streptomycin
2. Local—elevate the scrotum (adhesive bridge).
3. Apply hot compresses or ice packs—either works satisfactorily although heat gives more relief of the pain.

Acute epididymitis is usually followed by resolution, provided it is not tuberculous in origin.

Nonspecific Epididymitis.—This lesion may occur without determinable cause, presumably due to lowered tissue resistance. The epididymis becomes enlarged, hard, and tender. There is no urethral discharge, and frequently no history of exposure.

Treatment —

1. Penicillin or streptomycin should be given. Either will reduce the fever, but will not alter the eventual course of the disease.

2. Elevation of the scrotum and applications of heat or cold.

3. Incision and drainage of the testicle. If the epididymis is incised, pus is nearly always found, and varies in amount from a few drops to a half ounce. Incision and drainage usually give prompt relief of pain but do not otherwise influence the course of the disease. The acute infection may progress to abscess formation requiring drainage. Sterility of the affected testicle usually follows an epididymitis, probably due to obstruction.

Tuberculous Epididymitis (see page 567)

Syphilitic Epididymitis—very rare—(syphilitic orchitis in the latent stage of the disease is not uncommon)

VAS

Injuries of the vas are uncommon. The vas may be severed or damaged in hernia operations, most commonly in children. Inflammations of the vas either gonorrheal, nonspecific, or tuberculous are more common and cause pain in the inguinal region. With the exception of tuberculosis, they tend to subside when the associated epididymitis present resolves under treatment. On rare occasions localized abscesses form and require drainage. Beading of the vas on palpation is a sign of previous inflammation.

VESSELS

1 **Injuries of the vessels** are uncommon, with the exception of injury to a varicocele which may cause rupture of some of the vessels. Extravasation of blood into the

scrotum occurs. This tends to be absorbed if the patient is placed at rest.

2. *Varicocele*.—A varicocele is that condition in which the veins of the pampiniform plexus have become varicose. The extent varies. It is usually associated with a lax pendulous scrotum. It is relatively common on the left side in young men, vary rare on the right side, and occasionally bilateral. It is described as "a bag of worms" because of the appearance on the surface of the scrotum. In older men it may be due to venous obstruction caused by the pressure of a renal tumor.

There are several theories as to its cause, none of which are entirely satisfactory. The two most common reasons given are (1) incompetence of the valves in the left spermatic vein; (2) the greater intravenous pressure in the left spermatic vein. The stagnation of blood may result in partial atrophy of the testicle.

Signs and Symptoms.—

1. Varicocele may be asymptomatic or the patient may complain of pain in the testicle. In a great many cases there is a psychoneurotic element as the patient is conscious of an inferiority rather than pain.

2. When the patient stands, the vessels of the pampiniform plexus become distended and readily palpable. On recumbency with elevation of the scrotum, the vessels empty.

Treatment.—

1. Support the scrotum with a suspensory belt. Cold baths are sometimes helpful.

2. Operation in selected cases. The majority of the veins are isolated from the vas, making certain to leave the artery of the vas and a few veins behind. The main group is divided and a 1½ inch length excised. Each end is tied with a transfixing ligature and then approximated by tying the ligatures together.

TESTICLE

1. *Contusions*.—Blows to the testicle are fairly common. The majority of such in-

juries are fortunately slight. Severe trauma will cause ecchymosis of the scrotum, acute pain, swelling of the testicle, and exquisite tenderness. When the acute episode subsides, atrophy may follow.

Treatment includes bed rest, elevation of the scrotum and ice-bags to the scrotum.

2. *Wounds of the Testicle*.—Stabbing wounds of the testicle are uncommon. Extensive lacerations of the scrotum with partial or complete loss of glandular tissue are more common in the accidents of industries and modern warfare. After treating shock, the area is thoroughly cleansed with green soap and water, debrided, and every effort made to preserve as much of the testicle as possible. Usually very little tissue can be saved as the tunica albuginea is extensively torn and the seminiferous tubules bulge through the rent.

3. *Inflammations.*—

(a) *Acute orchitis*, nonspecific, may occur as a rare complication of any generalized infection. It is characterized by a sharp rise of temperature, chills, headache, pronounced weakness, leukocytosis, pain and swelling of the testicle. The epididymis and spermatic cord may be involved.

Treatment consists of elevation of the scrotum and the application of heat. Incision and drainage may be required later, and occasionally orchiectomy. Even if drainage is not required, testicular atrophy usually ensues.

(b) *Orchitis of Mumps*.—Acute orchitis occurs as a complication of mumps in 20% of cases. The incidence in infants is much less. Testicular atrophy follows in over half the cases. It has been claimed by some that the atrophy is due to an acute inflammatory hydrocele, and that if this is drained during the acute stage, atrophy of the testicle will not ensue. These claims, however, do not appear to be true, as testicular biopsy clearly shows that an acute inflammatory reaction occurs within the substance of the testicle which explains the subsequent atrophy.

(c) *Chronic Orchitis*—

- 1 Tuberculosis.
2. Syphilis.

Clinically, syphilitic infections of the testicle are rare. Yet at autopsy, syphilitic fibrosis of the body of the testicle is so common that it can be said that syphilis attacks the testicle in preference to any other tissue, with the possible exception of the heart and aorta.

Latent syphilitic orchitis occurs frequently and often passes unnoticed.

Congenital syphilitic orchitis develops between the 3rd and 10th month of life, and results in diffuse fibrosis and atrophy (of the testicle).

Gumma of testis is a late manifestation of syphilis.

4 *Neuralgia of Testicle*.—The complaint of pain in the testicle, usually intermittent, gnawing in character, and frequently severe, is most difficult to treat. It may be caused by involvement of the genitofemoral nerve in scar tissue, hernia, prostatitis, a localized healed lesion in the testicle, or low ureteral stone.

5 *Torsion of the Testicle*.—Torsion of the testicle is in fact rotation or torsion of the spermatic cord which shuts off the blood supply to the testicle. Torsion can occur only if the epididymis is not attached to the posterior scrotal wall, and if there is a complete and high investment of the testis and epididymis by the tunica vaginalis. If the tunica vaginalis is attached high to the cord, contraction of the cremasteric muscle with its spiral fibers may cause complete rotation of the testicle, shutting off the blood supply. The torsion is usually anti-clockwise and nearly always intravaginal. The etiology is unknown.

Symptoms and Signs—

1. Sudden acute onset of pain in the testicle. This may be agonizing.
2. Elevation of the testicle in the scrotum.
3. Prehn's sign—elevation of the scrotum accentuates the pain.

4 If of long duration, there is swelling of the scrotum with redness of the overlying skin, and the patient has some elevation of temperature.

Treatment.—

1. If soon after the onset—try to reduce the torsion manually, by twisting the supported testis from within outward. If this increases the pain, twist it in the opposite direction.

2. Up to 24 hours after onset—expose the testicle through an inguinal incision, untwist it, return it to the scrotum and suture it there.

3 If the torsion has existed longer than 24 hours, the testis will appear blue-black and lifeless, in which case it should be removed.

6 *Cryptorchidism* is congenital malposition of the testicle. It may be

- (a) Abdominal (nondescent).
- (b) Inguinal (partial descent).
- (c) Retention of the testicle in the upper part of the scrotum.
- (d) Perineal.
- (e) Penile.

Most cases encountered clinically are in the inguinal region, and due to incomplete descent. They are underdeveloped and are frequently subjected to repeated trauma. For years it has been claimed that the incidence of malignancy in an undescended testicle is exceedingly high as compared to a normally descended testicle. Recent work would indicate that this is not true and that the incidence of malignancy is no higher than in a normally descended testis. Atrophy of the testicle with aspermatogenesis is a frequent finding.

Treatment for Incomplete Descent of the Testicle (Cryptorchidism).—The ideal age for treatment is up to 9 to 11 years.

1 *Hormonal*: In some children the testes are either high in the scrotum or in the inguinal canal. In some of these, the injection of 500 units of Anterior Pituitary-Like Substance (A.P.L.) daily for 20 doses, followed by a rest period of 10 days, and then

another series of 10 injections will cause the testes to descend. In many cases, however, the testicle is bound down to the surrounding tissue by adhesions. In these A.P.L. will not cause the testicle to descend.

In practice, it is customary to give a course of A.P.L. prior to proceeding with surgery. If the testicle does not descend, and surgery is required, it is found that the tissues are more pliable after a course of A.P.L., and the testicle has an increased blood supply.

2. Surgical: If hormonal therapy fails to bring the testicle down into the scrotum, the inguinal canal should be opened, the cord and testicle found, and the congenital hernia repaired. The cord can then be lengthened sufficiently to allow positioning of the testicle in the new bed made for it in the scrotum.

Tumors of the Testicle

The views held regarding tumors of the testicle have altered considerably during the past twenty years. In 1929 Zondek discovered Prolan A in the urine of a patient suffering from teratoma testis. Soon it became common practice to regard the quantitative amount of Prolan A found in the urine as an index of the type of tumor present, and the prognosis. Opinion has now shifted so that it may be said that whereas a positive Prolan A in the urine indicates the presence of a testicular tumor, a negative Prolan A does not exclude a tumor. In chorionepithelioma there is a very high excretion of Prolan A.

Classification

| <i>Tumors of the Testicle</i> | <i>Incidence</i> |
|--------------------------------|------------------|
| 1. Seminoma (germinoma) | 35% |
| 2 Teratoma | 42% |
| 3 Embryonal Carcinoma | 23% |
| (including chorionepithelioma) | |

Incidence.—Tumors of the testicle are uncommon, but occur with sufficient frequency to be important. They form approximately 20% of all tumors in the male,

and are commonest between the ages of 20 and 40 years. Testicular neoplasms may be found at any age, even in the newborn, and most clinicians and pathologists agree that they are all malignant or potentially so.

1. Seminoma (Synonyms; *germinoma*; or *embryonal carcinoma with lymphoid stroma*).—One-third of all testicular tumors are monocellular seminomas made up of rounded polyhedral elements with sharp cell borders. The cytoplasm is often clear. The cells are usually arranged in unorganized masses of cords divided by trabeculae of connective tissue. Variations in structure may occur. The lymphocytic infiltration of the connective tissue trabeculae in seminomas has led some clinics to use the term *embryonal carcinoma, with lymphoid stroma*. This is considered to be a misleading term. Foci of necrosis are often encountered. Invasion of the adnexae and cord is exceptional while in embryonal carcinomas it is the rule.

The resemblance between seminoma cells and some of the seminiferous tubules is taken as evidence that seminomas arise from spermatogonia cells of the seminiferous tubules.

2. Teratoma.—There is a great range of variation in the structure of teratomas. If there are no recognizable histological components, the term *adult* may be used. Such are never benign, because metastases of testicular tumors which appeared to be only adult teratomas have been known.

Teratomas are characterized by the presence of epithelial masses, glands, and cysts. Many of these structures are organized in combination with undifferentiated or specialized mesenchymal tissues such as cartilage. Transitions occur and the resulting picture is one of mixed figures in which some semblance of organization can often be discerned. Squamous cell nests and cysts, often keratinizing, are frequent; glandular appendages and adult hair are rare. Epidermoid cysts outnumber dermoid cysts 10:1.

3. Embryonal Carcinoma.—(A) These tumors which include embryonal adenocarcinoma and papillary adenocarcinoma are of-

ten confused with seminomas, although they differ not only in fundamental cell type but in biological behavior and prognosis as well. They show considerable variation in cell type, e.g., epithelial cells, cuboidal cells, columnar cells and papillary structures. The cell type is of no known biologic significance and does not determine the prognosis.

(B) *Chorionepithelioma*.—These tumors may be suspected grossly because they are strikingly hemorrhagic. In addition they produce a very high excretion of Prolan A in the urine (100,000 or more mouse units per liter).

Microscopically they are easily recognized because of their characteristic appearance. They have two cellular components arranged so as to duplicate the architecture of the placental villi. The combination of compactly grouped cyto-trophoblastic cells and giant multi-nucleated syncytial structures, arranged at the borders of the cellular masses, makes one of the most striking microscopic pictures in all pathology.

Signs and Symptoms of Tumor of the Testicle.—Tumor of the testicle may occur at any age, but 80% occur between the ages of 20 and 40 years. There are two cardinal symptoms:

- 1 A painless swelling in the testicle
- 2 Sense of weight in the scrotum. Attention is often drawn to the testicle by some insignificant trauma, or the patient may have noticed a gradual enlargement to which at first he paid little attention.
- 3 The first symptom noted may be the result of metastases or of lymphatic involvement, e.g., loss of weight, abdominal pain, pain in the back or spine or a slight cough.
4. On palpation, the testicle is usually smooth and regular in early cases, but nodular and cystic in later cases. The skin is red and shiny from stretching, the surface blood vessels are dilated, and at a very late stage, ulceration through the scrotum may occur.

5. The presence of Prolan A in the urine, i.e., a positive Aschheim-Zondek test, is in-

dicative of tumor being present in the testicle, but a negative test does not exclude tumor.

Differential Diagnosis.—

1. Tumor
- 2 Hydrocele—(often present with tumor)
- 3 Hematocele.
- 4 Tuberculosis.
5. Syphilis

All testicular swellings must be regarded as malignant until proved otherwise.

Metastases.—Metastases occur via the lymphatics to the deep abdominal and aortic glands, and via the blood stream to give widespread distal metastases. The lungs are most commonly involved. Blood stream invasion may occur early.

Prognosis.—The prognosis in testicular tumors is very poor. There is a 1 to 5 year cure in many cases of seminoma. So far, there has been no case of chorionepithelioma known to be cured. The prognosis depends in part upon:

- 1 The extent and rapidity with which the tumor grows and metastasizes.
- 2 The radiosensitivity of the tumor, e.g., abdominal metastases sometimes appear to melt away (temporarily) following radiation of the abdomen.

Treatment.—In the treatment of testicular tumor, one of several courses may be pursued as follows.

- 1 Simple orchiectomy.
- 2 Orchiectomy and irradiation
- 3 Radical operation with removal of the regional and retroperitoneal lymph nodes on the affected side, followed by deep x-ray irradiation
4. Irradiation alone
- 5 Bilateral orchiectomy

REFERENCES

- Lowsley, O. S., and Kirwin, T. J.: *Clinical Urology*, ed. 2, Baltimore, 1944, The Williams & Wilkins Company.
- Rolnick, H. G.: *Practice of Urology*, in 2 volumes, Philadelphia, 1949, J. B. Lippincott Company.
- Wimbury-White, H. P., editor: *Textbook of Genito-Urinary Surgery*, Edinburgh, 1918, E. & S. Livingstone.

CHAPTER XXXII

PERIPHERAL VASCULAR DISEASES

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CONGENITAL VASCULAR ANOMALIES

- I. Hemangioma
- II. Congenital Arteriovenous Fistula
- III. Glomus Tumor

I. Hemangioma

This group of tumors consists of an actual new formation of blood vessels, probably arising from isolated elements of the vascular mesenchyma which have not been included in the formation of the normal vascular tree. The evidence for this theory is the relative frequency of circumscribed angiomas with one to three entering arteries and an equally small number of efferent channels. The extensiveness of the hemangioma is probably an indication of its date of commencement, the small ones commencing toward the end of fetal life, while the extensive ones involving superficial and deep tissues arise early in the development of the embryo. The tumor usually continues to enlarge until growth ceases, but in those where arteriovenous connections are present, such as the cirroid type, the increase in size is continuous. Pregnancy and the onset of menstruation may result in an increase in the rate of growth.

Most of the hemangiomas are present at birth and most of the remainder appear in early childhood. It is the commonest tumor of infancy and childhood and, for some unknown reason, is twice as common in females as in males.

A. CAPILLARY HEMANGIOMA

In this group are several varieties which have acquired different names, but all are similar in respect to their congenital origin

and the fact that they occur in various surfaces, chiefly the skin and mucous membranes. The blood vessels are capillary in type, lined by a single layer of endothelium and are sparsely filled with blood.

Port Wine Stain.—This deforming manifestation of a capillary hemangioma appears at birth and grows with the development of the child. The condition involves the derma and exhibits diffuse telangiectasia but no tumorous enlargement. With years, the color tends to change from a pink to a bluish purple hue. The face and oral cavity are most frequently involved but the trunk and limbs are also commonly affected.

Treatment is difficult and no one method is apt to give a good result. The abrasive method of Jonsson is probably the best available. In this, the involved area is rubbed with sandpaper to destroy the cutis and the capillary dilatations. Care must be taken to leave part of the derma, as scarring will otherwise result. Novocain with adrenalin is used as the anesthetic to produce insensitvity and reduce hemorrhage. A moderately large area can be treated in one sitting. Tattooing of the abnormal area is also used, employing a mixture of pigments to produce as normal a skin color as possible. Only small areas can be dealt with at one time and consequently treatment may take months. The cosmetic called "Cover-Mark" is an excellent way of camouflaging this deformity.

Spider Nevus is another variety of capillary hemangioma, but is distinctive because of its small size and the appearance of a central red spot with numerous radiating fine blood vessels (spider legs). The central red spot is a small artery which can be

shown to pulsate with a rhythmic flush and pallor corresponding with the pulse beat, but having a much lower pressure than that of the arterial pressure. The face, trunk and upper extremities are most commonly involved, but they also occur in the mucous membranes of the mouth and nose.

These spider nevi develop in relationship with several apparently unrelated conditions. Their connection with liver disease has been known for almost a century. Especially in cirrhosis, these nevi appear and disappear with the exacerbations and remission of the disease. The reason for this association is as yet pure speculation. Pregnancy is another condition which predisposes to spider nevi, increasing in size and number until parturition when they completely disappear. The appearance of these lesions on the nose is often the telltale mark of a chronic alcoholic.



Fig. 253—Capillary hemangioma of the port wine stain type associated with several strawberry type hemangiomas.

Treatment, when indicated, is simply done by touching the central spot with a pointed electrode using unipolar coagulating current.

Strawberry Mark is one of the most common varieties of capillary hemangioma and is of congenital origin even though its appearance may be delayed. This variety is a sessile lobulated circumscribed bright red tumor and appears most commonly on the head and face. They vary in size from that of a pea to over an inch in diameter. The blood vessels are in masses but are capillary in type and contain only a small quantity of abnormal red blood cells.

Treatment of this variety of hemangioma is most effective by the use of local freezing. Pencils of "dry ice" (CO_2 ice) are made and locally applied for ten to twenty seconds and repeated every two weeks for as many applications as necessary. Thrombosis of the vessels takes place with late organization and fibrosis. X-ray or radium may be used but with less success and with the possible later development of telangiectasia in the radiated skin. Surgical excision of the smaller types is most effective and leaves minimal deformity.

Hereditary Hemorrhagic Telangiectasia (Rendu-Osler-Weber's disease). This variety of hereditary capillary abnormality apparently consists of a localized maldevelopment of minute blood vessels with a predisposition to rupture and hemorrhage. The nose is the commonest area involved and many severe epistaxes are from this cause. A familial history is present and the lesion consists of one or more small angiomas either in the nose or mouth. They appear as tiny red or purplish dots, punctate, sessile or spider-like. The tumor may appear in childhood but severe hemorrhages are usually seen in middle life. The condition should be kept in mind in major nose or oral bleeding and treatment consists of electrical fulguration or the local application of radium.

B. CAVERNOUS HEMANGIOMA

In this variety of angioma the blood vessel spaces are much larger than the capillary type. This is apparently due to an increased connection with the systemic circulation with the formation of vascular sinuses and sacculations set in a reticular network. This type is also congenital but may not be noticed until later in life when the gradual enlargement becomes manifest. They are soft and readily compressible and, when present in the subcutaneous tissues, reveal themselves as an irregular lobulated spongy mass with occasional bluish areas seen through the skin. No pulsations or bruits can be detected. Besides the superficial structures, these benign tumors are found in the abdominal organs, muscle, and bone. The liver is the most common abdominal organ involved and the vertebra the commonest osseous structure.

Treatment of the superficial variety is best accomplished by the injection of sclerosing fluids into the vascular channels. Thrombosis, organization, and fibrosis of the tumor result. Surgical excision is also successful in the superficial types. Deep x-ray therapy is the only treatment at present useful in the deep types.

C. RACEMOSE OR CIRROID ANEURYSMAL HEMANGIOMA

The clinical appearance of this variety of angioma is that of a pulsating mass of tortuous vessels. They are found most commonly on the head, face or neck and may arise relatively suddenly from a pre-existing angioma or more gradually without apparent cause. These are true localized arteriovenous fistulas and cause pressure effects by their enlargement. When on the head, a bruit can be heard as well as felt, and bony erosion of the skull is usually present. When in the orbit, marked proptosis takes place. They are usually directly connected with one or more major branches of the carotid system which require ligation before satisfactory therapy can be carried out.

Surgical excision may be feasible in the smaller varieties after ligation of the feeding arteries or, if complete removal is impossible, preliminary arterial ligation followed by injection of sclerosing fluids or radiation therapy frequently produces satisfactory results.

D. HEMANGIOSARCOMA

In this group are included the sarcomas originating from vascular tissue and consisting of marked new formation of blood vessel spaces and vascular channels. The spaces are highly irregular and follow no set pattern. They are difficult to distinguish from highly vascular sarcomas originating in other mesodermal tissues. As can be imagined, the degree of malignancy is high and metastases occur early via the hematogenous route. Clinically, the surface types resemble a cavernous hemangioma but, due to their extreme vascularity, may show pulsation and a bruit. The prognosis is invariably bad, although deep x-ray therapy has a restraining influence on the local spread.

II. Congenital Arteriovenous Fistulas

In the development of the vascular system in the early embryo, the arteries and veins develop from a common vascular tube.



Fig 254—Localized congenital arteriovenous fistula involving the pinna.

Certain parts of this tube become the arterial and the other parts the venous component. This helps to explain the rather rare condition of congenital arteriovenous fistula where (usually multiple) direct communications exist between otherwise normal arteries and veins. Various varieties occur, the localized ones resembling the cavernous

hemangiomas and the generalized variety revealing their presence by generalized changes in the extremity.

Localizing Types.—The terms cirroid and racemose aneurysms are used in this variety, the latter applying to those in the neck. The points of differentiation from a cavernous hemangioma are the greater extent of the lesion, more rapid progression, the pulsatile character and the presence of a bruit over the lesion. Frequently in association are other vascular anomalies and abnormalities such as capillary hemangiomas and marked engorgement of superficial veins.

Generalized Types.—As mentioned previously, the extremities are most commonly involved in this variety and the diagnosis is made by the recognition of the effects of these vascular communications. Varicose veins, due to increased venous pressure, are almost invariably present coming on early in life and being gradually progressive. Consequently in a young patient with severe varicose veins, the condition should be especially suspected. The complications of these veins such as ulceration and eczema may first bring the patient to medical attention. There is an associated enlargement of the limb because of the venous engorgement, increased length of the leg because of epiphyseal vascular stimulation and increased warmth. Increased sweating and growth of hair are also commonly seen. Various types of surface hemangioma may be associated. A bruit or thrill is rarely present due to the small size of the fistulas.

Laboratory aids in arriving at a diagnosis are the finding of increased oxygen values in the venous blood and arteriography will indicate the multiple intercommunications. Treatment is unsatisfactory due to the numerous small fistulas making impossible their interruption. Excision or stripping of the dilated superficial venous channels is indicated especially where complications as the result of these veins are present. Compression bandages or strong elastic stocking, to give an increased tissue tension in-



Fig. 255.—Generalized type of congenital arteriovenous fistula involving the palm and fourth finger. Note the marked enlargement of the forearm veins.

hibiting the arteriovenous shunt, should be worn continuously. A scrupulous search should be made over the area with a stethoscope to determine areas of localized bruits. Such a bruit indicates a collection of large arteriovenous shunts which, being localized, are capable of surgical excision.

III. Glomus Tumor

Glomus tumors are not definitely congenital in origin but are included in this group for convenience in classification. The glomus body is a specialized arteriovenous communication which develops shortly after birth and which has the function of local and general heat regulation and also some connection in the regulation of blood pressure. These glomus bodies are microscopic in size and are most frequently found in the extremities but are also present in other parts of the body. As many as seventy such bodies may be present in one toe. They consist of thickened arteriovenous vascular channels associated with large epithelioid cells which are peculiar to these bodies and are known as glomus cells. Numerous non-myelinated nerve fibers are in close association.

Glomus tumors are overgrowths of the glomus body enlarging to macroscopic size. A common situation is beneath the nail but they are also present in other locations in the extremities. The enlargement is slow and metastases do not take place. Recurrence has occurred following excision, but such a recurrence may be due to the development of a new tumor in a contiguous area.

The diagnosis is not difficult if the condition is kept in mind. They manifest themselves as small pinhead or slightly larger bluish nodules in the skin or beneath a nail. Pain is the predominant symptom. It is neuralgic in type, present in the region of the tumor, and is aggravated by cold or heat. Local pressure on the spot produces severe, excruciating and lancinating pain to such a degree in some cases that the patient shrinks from the slightest contact. When

beneath the nail, erosion of the underlying phalanx may take place by direct pressure of the tumor. Treatment consists of surgical excision.



Fig. 256—Generalized congenital arteriovenous fistula involving the left lower leg. Note the superficial vein enlargement, atrophic changes in the foot, and the increased leg length.

TRAUMATIC ARTERIAL LESIONS

Arterial Spasm

The smooth muscle of the arterial wall has the property of local contraction of a small segment or, as the result of irritation of a localized area, of producing an arterial spasm of the entire limb. Whether the mechanism of this vessel contraction is the result of local overstretching of the smooth muscle fibers and consequent spasm or whether the injury causes severe sympathetic stimulation is not certain. The fact remains that subsequent to trauma of many kinds, arterial spasm can occur severe enough at times to cause complete ischemia of the limb and subsequent gangrene.

This condition is seen most often in wartime due to the passage of high velocity missiles close to major arteries with momentary lateral stretch of the vessel. It has followed other soft tissue wounds, the reduction of fractures, various orthopedic operations, and has been reported as following the injection of sclerosing fluids in the treatment of varicose veins. All degrees of spasm occur from the minor unimportant transient degrees to the severe type with resultant gangrene. The condition known as Volkmann's ischemic contracture of the forearm and hand following elbow injuries and fractures is probably due to this mechanism.

The prognosis of the limb in the severe states depends on early recognition of the condition in the acute phase so that gangrene can be forestalled. The color, temperature and sensation of the hand and foot along with the peripheral pulses should be palpated routinely after every limb operation or injury because the majority of extremities lost from this cause are the result of late recognition. Dressings should be loosened, splints removed, and abnormal stretching of joints released. Antispasmodic drugs such as Priscoline 50 mg doses intravenously are of some value, release of sympathetic tone by procaine block of the appropriate sympathetic chain should be done

Increased body warmth and the correction of anemia and blood volume are also indicated. If conservative treatment fails, then surgical exposure of the affected artery is indicated with possible excision of the spastic segment.

Arterial Contusion

Damage to a segment of arterial wall can be produced by direct violence of the original traumatic agent, by operative trauma or by damage to the artery by a fractured segment of bone. The result of this trauma is hemorrhage into the arterial wall leading to possible thrombosis because of the intimal damage or secondary hemorrhage from later rupture of the damaged artery. As indicated above, possible widespread arterial spasm may also be associated.

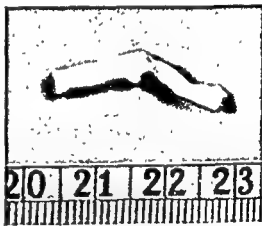


Fig 257—Section of brachial artery removed from case of severe fracture of the humerus showing arterial contusion

Treatment is only indicated when total ischemia is present or threatened, and it is only when the artery is exposed surgically that the lesion can be differentiated from arterial spasm. Arterectomy of the damaged segment is indicated.

Partial Division with False Aneurysm

When a major artery is partially severed, especially if done by a small sharp object such as an icepick, fragment of a grenade

shell or occasionally a bone spicule, bleeding occurs from the arterial hole into the surrounding tissues but is prevented from escaping to the exterior by the smallness of the missile puncture wound. A deep hematoma develops with thrombosis and fibrosis of its outer layers and containing fluid blood in its interior which flows to and from the arterial tear. The lesion is also aptly called a pulsating hematoma and is a false aneurysm inasmuch as the cavity has no endothelial lining.

The diagnosis of such a lesion depends on a careful search in all puncture wounds and fractures for the following signs:

1. A pulsating swelling in the region of injury.

correction is indicated with removal of the false sac (if possible) and closure of the arterial tear.

Complete Division

This type of arterial trauma is the most serious of all both from its high mortality and high incidence of gangrene of the limb. If the wound is open, death from exsanguination is probable unless rapid first-aid measures are carried out. If the external wound is small or if produced by a closed type of injury, then a massive hematoma rapidly collects.

In either instance, the distal parts are threatened with gangrene due to partial or complete ischemia, the degree of ischemia

Incidence of Gangrene in Main Vessel Interruption

| | Ligation Only | World War I wounding + ligation | World War II wounding + ligation |
|------------|------------------|------------------------------------|-------------------------------------|
| Subclavian | 0 | 8.8% | 24.0% |
| Axillary | 1.4% | 2.7% | 28.0% |
| Brachial | 0 | 4.0% | ? |
| Femoral | 17.2% | 20.2% | 32.0% |
| Popliteal | 26.6% | 34.7% | 86.0% |

Fig. 258

2. A systolic bruit and thrill over the involved area, the bruit being propagated distally.

3. Later evidence of nerve compression by the hematoma which may increase gradually in size.

It is only in the most major degrees of arterial puncture and partial division that the distal circulation is impaired.

Initial treatment consists of immobilization of the limb and pressure over the injured part. If the pulsating tumor is not increasing in size and evidence of progressive nerve pressure is absent, then conservative therapy is indicated for a time because the smaller varieties tend to heal spontaneously. If persisting beyond three months, surgical

paralleling the amount of remaining functioning collateral vessels.

Rapid surgical measures are indicated consisting of replacement of the blood loss and exposure of the severed artery. Ideally, débridement of the severed arterial ends

Late Results Following Arterial Ligation

Muscles:

1. intermittent claudication
2. nocturnal cramps (rest pain)
3. paralyzed extensor hallucis longus muscle
4. paralysis of small muscles of hands and feet

Sensory:

1. cold dead feeling of limb
2. anaesthesia of glove or stocking type
3. hyperalgesia
4. delayed pain response to noxious stimuli

Fig. 259.

should be done with end-to-end suture. However, in the majority of cases this is not feasible because of the extent of the arterial damage. The majority of such cases will require ligation of the damaged artery with subsequent all-out efforts to keep the functioning collateral vessels as dilated as possible. This is accomplished by repeated lumbar sympathetic procaine blocks or sympathectomy if the patient's condition will allow. However, an attempt should be made to bridge the arterial gap by a segment of vein taken from the saphenous or superficial femoral vein because of the high incidence of gangrene following ligation. The greatest number of these cases are seen during warfare and it is likely that during World War III, banks of homogenous arterial grafts will be available to the forward surgeon for this type of case.

In either instance, anticoagulant therapy should be given for 8 to 10 days postoperatively, in the first instance to prevent thrombosis in the distal segment of the ligated artery and its collaterals, and in the grafted case to keep the anastomosis patent.

Arteriovenous Fistula

A small sharp object such as a fragment of shell may puncture both the main artery and vein. In such an instance the usual false aneurysm does not occur because the escaping arterial blood flows directly into the vein. Such fistulas rarely close spontaneously and soon become lined by vascular endothelium. Due to the pouring of arterial blood directly into the venous system through a relatively large opening, marked abnormal physiological changes result which affect the entire cardiovascular system.

1. An immediate fall in blood pressure which later returns to normal levels, but a permanently lowered diastolic pressure remains giving an increased pulse pressure.

2. Increase in pulse rate. This increased rate is abruptly slowed by 11 to 16 beats per minute (depending on the size of the fistula) when the artery is compressed proximal to

the fistula. This phenomenon is known as Branham's sign.

3. Increased venous pressure in the veins both distal and proximal to the fistula due to the direct dumping of arterial blood into the vein.

4. Increased cardiac output due to increased inflow.

5. A gradual increase in total blood volume.

6. A gradual dilatation and hypertrophy of the heart which may go on to cardiac failure when the fistula has been present for long periods.

Clinical manifestations include a thrill over the fistulous tract with a bruit which is loudest in systole but is also heard in diastole (so-called machinery murmur). Decreased peripheral arterial flow will be evident depending on the size of the fistula. Diminished distal arterial pulses will be present and the ischemia may be severe enough in the large fistulas that distal gangrene may develop. The size of the extremity is increased and also elevated surface temperature is present. In later cases, evidence of increased venous pressure will be present in the form of varicose veins, some dependent cyanosis, edema, and possibly the complications of venous stasis (ulceration and eczema). An arteriogram will give the location and size of the fistula.

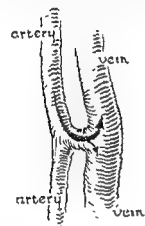
Treatment to close the fistulous tract is indicated because of the late cardiovascular changes. This should be delayed for more than two months to allow the collateral vessels time for maximal dilatation. A preliminary sympathectomy of the involved limb is also useful in this respect to minimize the dangers of acute arterial ischemia to the distal limb immediately after operation. The surgical operation of choice is repair of the arterial opening so that the continuity of the main artery is preserved. However, this is frequently not feasible and the operation most commonly employed is quadruple ligation, tying the artery and vein both proximal and distal to the fistula with, preferably, excision of the fistulous area.

ARTERIAL DISEASES

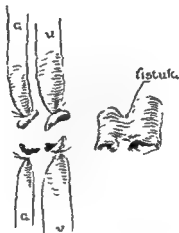
Thromboangiitis Obliterans

Thromboangiitis obliterans was first described as a definite entity by Leo Buerger in 1908 and so is commonly known as Buerger's disease. It had been previously

disease can be defined as a segmental, inflammatory, obliterative disease of the arteries and veins which occurs almost exclusively in young men involving the extremities and occasionally the viscera. The Jewish race appears more susceptible than the Gentile for reasons which are unknown.



diagrammatic sketch
of traumatic
arterio-venous
fistula



quadruple ligation
with excision
of
fistulous tract



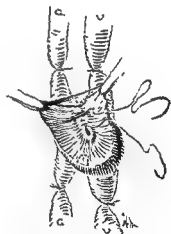
aneurismorrhaphy
with reconstruction
of artery—



proximal & distal
ligation
of artery—



ligation of
fistulous tract



quadruple ligation
with obliterative
endoaneurismorrhaphy

Fig. 260.—Types of operations on arteriovenous fistulas.

described, but with little differentiation from the other types of occlusive arterial diseases. Buerger's pathological studies made him conclude that the condition was a subacute inflammation of the vessels leading to thrombosis with subsequent organization and partial recanalization of the thrombus. The

Etiology.—The exact cause of this disease is uncertain. Various theories have appeared but each is incomplete. These include focal infection; an allergic response to unknown stimuli and hence related to periarteritis nodosa and the collagen diseases, and finally, tobacco. This latter factor is

the only one concerning which definite knowledge exists and it appears to be true that the disease is adversely affected by smoking. The causative factor in tobacco smoke is as yet unknown as it has been shown that denicotinized tobacco is just as harmful as the true product.

The high preponderance of this disease in males suggests also some protective influence in the female. This is possibly the vasodilating effect of the estrogens or some other side effect because experimental evidence has shown that female rats are less likely to get gangrene of the tail and that male rats are protected from the effect of ergotamine injections when given theelin. The unprotected male rat is invariably affected following ergotamine.

Pathology.—The disease varies in intensity in different individuals and may be a slowly progressive one with long periods of quiescence or may be a rapidly fulminating type with rapid progression and early loss of one or more limbs due to gangrene. The disease can be divided into four types according to severity:

1 Nonprogressive type in which a good collateral circulation develops and which becomes quiescent after one or two episodes

2. The type presenting sudden major arterial or venous occlusion

3 Slowly progressive type which may eventually lead to gangrene or leave a badly impaired arterial tree.

4 Fulminating rapidly progressive type requiring multiple amputations

Microscopically, one sees a diffusely cellular thrombus blocking the arterial lumen, affecting chiefly the smaller arteries such as the dorsalis pedis, posterior tibial, ulnar, etc. The arterioles and the large arterial trunks are seldom involved. A well-preserved media is present and there is some degree of endothelial proliferation. Fibrosis and lymphocytic infiltration of all coats is present especially in the adventitia and about the vasa vasorum. The accompanying nerve is

frequently involved in this periarterial fibrosis and the vein can show the same involvement of the wall as is seen in the artery. It appears as if some factor primarily involves the endothelium causing a proliferative and irritative effect which results in exudative changes and thrombosis. It is possible that the fibrosis is a reparative aftereffect. Calcification in the vessel wall is very rare.

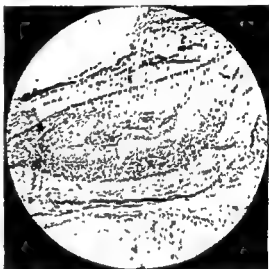


Fig 261—Low power photomicrograph showing arterial occlusion and partial recanalization (Buerger's disease)

Diagnosis.—It must be emphasized that this disease occurs between the age limits of 15 to 45 years, and it is very doubtful whether such a diagnosis should be made in a person whose symptoms commenced after the age of 45 years. The condition itself is silent and manifests itself only from the effects of reduction in blood supply. Therefore suspect thromboangiitis obliterans in any case of intermittent claudication, unexplained pains or sensory changes in the legs, ulcers that refuse to heal and gangrene of any degree when in the age group mentioned above. Such a person will almost invariably be a male, possibly Jewish, and a smoker. About 30% of cases show evidence of involvement of the upper as well as the lower extremities.

The signs of arterial ischemia include thinning of the skin of the foot, with shininess, loss of elasticity, loss of subcutaneous fat and trophic changes in the nails. Blanching of the foot on elevation and rubor on dependency will be seen to a degree depending on the extent of the arterial ischemia. The more involved foot will be cooler

in the arms as well as the legs, thromboangiitis is almost invariably the cause. In association with the arterial involvement, about one case in four will show evidence or give a history of repeated episodes of superficial vein phlebitis occurring in small segments of previously normal veins.

Treatment.—Because no specific etiological factor is known, treatment is mainly directed toward an effort to increase the blood flow in the functioning collateral vessels. But, first of all, further smoking should be strictly prohibited because it is the opinion of the majority of observers that this disease will continue to progress as long as the patient smokes



Fig 262.—Buerger's disease involving the right hand with superficial gangrene of the tip of the third finger



Fig 263.—Photograph of a foot with Buerger's disease and gangrene of the big toe.

than the opposite one, and indolent ulceration or various degrees of gangrene may be present. The pain associated with the ulceration and gangrene is usually severe, much more so than in the lesions of arteriosclerosis obliterans. Palpation will reveal loss of pulsation of one or all of the main vessels of the limb; this can be confirmed by the oscillometric readings which will be less than the normal minimum of 1° in the calf. However, all that the above clinical data will demonstrate is that arterial occlusion is present; biopsy of a segment of involved artery is the only certain means of arriving at a diagnosis. But on clinical examination, when signs and symptoms are present

Means of increasing the blood flow in the collateral vessels can be of various types.

1. Buerger's exercises or the oscillating bed where by rhythmic elevation and lowering of the limb, blood flow is aided by gravity in its emptying and filling.

2. Alcohol by mouth because of its vasodilating and analgesic properties.

3 Reflex vasodilatation achieved by heating the trunk or upper extremities (where the result is desired in the lower extremity) to 115° F. for 15 to 20 minutes 3 or 4 times a day

4 Vasodilatation the result of artificially induced fever, the most common method being the use of typhoid fever vaccine which when given intravenously in the appropriate dose produces a chill and temporary fever.

5 Anticoagulant drugs, usually Dicumerol, to prevent the propagation of the thrombosis. This mode of therapy has only a limited usefulness, as hospitalization is necessary for proper control of the prothrombin level

6 Vasodilatation produced by drugs such as Hydergine (Sandoz) and Priscoline (Ciba), which act on the effector mechanism in the vessel wall inhibiting the vasoconstrictive effect of Adrenalin, are of limited usefulness but worthy of a trial in some cases

7. Extreme general care of the extremities to prevent any break in the skin and so allow infection to gain entrance to this devitalized tissue. It can be seen that any minor operation on such a hand or foot is contraindicated unless a careful assessment of the circulatory status has been made. It is a sad commentary on medical teaching that this point is so frequently neglected and that the doctor himself often initiates the gangrene by a misguided local operation

8. The best means of producing permanent vasodilatation is by interruption of the appropriate sympathetic supply to the limb. A sympathectomy will produce the maximal therapeutic dilatation possible, and is indicated in most cases where it can be shown by a preliminary procaine block of the appropriate sympathetic chain that vasodilatation of the collaterals is still possible

9. In the treatment of a local area of ulceration or gangrene, dry dressings only are indicated to avoid maceration of the skin. Amputations of digits, part of the foot or

part of the leg are relatively common necessities in Buerger's disease where conservative measures have failed.

ARTERIOSCLEROSIS OBLITERANS

Arteriosclerosis is a result of aging and in consequence is present eventually in all older people and is the greatest single cause of death. It involves the entire vascular tree, but the greatest involvement is in the major vessels. However, it is not the arteriosclerosis itself which is the lethal factor, but the results of this change. A severely arteriosclerotic artery can carry enough blood to satisfy the needs of a limb, and it is only when it becomes narrowed or blocked by atheromas or thrombus formation that symptoms occur from ischemia. The same applies to other more important arteries such as the coronary or cerebral vessels.

Pathology—The changes in arteriosclerosis are the result of the wear and tear phenomena taking place throughout life. However, in certain individuals and in certain disease states, arteriosclerosis appears to develop earlier than in the majority. Calcified spots in major arteries are not uncommon in the late thirties, and arteriosclerosis appears earlier in individuals with diabetes or other lipid metabolic upsets such as xanthoma tuberosum and myxedema. The increased blood cholesterol appears to be an influencing factor

Two main pathological types of arteriosclerosis are seen, the more common being the subintimal degeneration and thickening which goes on to produce atheromatous ulcers projecting into the vessel lumen or which become calcified to form the typical plaque. Fibrosis of the media and adventitia is also associated and the elastic laminae show degeneration. The above changes account for the rigidity and tortuosity which an arteriosclerotic vessel demonstrates. Calcification may be present not only in the plaques but may be diffusely laid down in the area between the media and intima

With such changes therefore in all the coats of the vessel especially the intima, it is not surprising that thrombus formation takes place in the lumen. A second variety of arteriosclerotic change is that frequently seen particularly in the arteries of the extremities. This consists of necrosis and calcification of the media, but without gross

material. Frequently the two processes are combined. This is most likely in vessels where atheromatous ulceration is present. Such ulcers are commonly situated in large arteries such as the lower end of the aorta where the volume and force of the blood flow prevent for a long time a complete occlusion. Blockage may occur in smaller



Fig 264—Diabetic arteriosclerotic gangrene involving the right great toe. The trophic ischemic changes in the toes and foot are well shown

internal change and again the vessels become transformed into rigid tubes (Monckeberg type).

The important phase in the production of arterial obliteration is the development of a thrombus in the vessel lumen or the occlusion of the vessel by masses of atheromatous

vessels where the changes are minimal and no evidence of calcification is seen on x-ray.

Symptoms and Signs in the Extremities.—

It is a fact that the arteries of the upper extremity are rarely involved from the clinical standpoint by arteriosclerosis obliterans and, if one artery does become blocked, the col-

lateral circulation is sufficiently good to prevent distal ischemic changes. Therefore, it is the lower extremities which almost invariably suffer from this disease. The patient seeks medical attention usually because of the sequelae of inadequate arterial inflow to the limb, and it must be emphasized that these symptoms are not typical of arteriosclerosis obliterans alone but are present in arterial ischemia due to any cause. The most important symptom is pain which is of two main types, the so-called rest pain where malnourished tissue produces a scalding, burning pain probably the result of ischemic changes in the somatic nerves. This pain is always worse at night when the legs are horizontal, aggravated by elevation of the limb and relieved by hanging the legs over the side of the bed. Pain is also present in association with gangrenous and ulcerative lesions. The second main variety of pain is the type known as intermittent claudication. This is a most exact and diagnostic symptom and, as before mentioned, can occur in any variety of arterial obliterative disease of the limb. Intermittent claudication is a cramplike, squeezing pain occurring most commonly in the calf muscles on exercise. The location of the pain depends on the level of the arterial blockage. It may be felt in the foot when the occlusion is in the region of the knee, in the calf when the block is in the thigh or in the lateral thigh when the iliac or common femoral artery is involved. This pain is due to muscle ischemia and therefore is felt only on exercise. Rapid walking, walking uphill or climbing stairs will initiate the pain sooner than a slow pace on level ground. After a rest of one to two minutes, the pain disappears only to appear again after a similar distance has been covered. The distance a patient can walk before the onset of intermittent claudication is a good index to the severity of the arterial occlusion and may vary from 50 feet in the severe cases to a quarter mile in the milder ones.

Other symptoms of arterial ischemia are coldness of the feet, nonhealing infections or

ulcers and various sensory changes including a hyperesthesia or a numbness and sensations of pins and needles. Examination shows evidence of varying degrees of trophic ischemic changes in the soft tissues of the foot such as thinning with shininess and loss of elasticity of the skin, decrease in the amount of subcutaneous tissue and atrophy, brittleness, and slow growth of the nails. Hair growth is poor and hair may be shed



Fig. 265—Technique of palpation of the dorsalis pedis, posterior tibial, and popliteal arteries.

from the affected extremity. Sweat glands atrophy and the foot appears dry and scaly. Ischemia on elevation occurs because of increased venous return and retarded arterial inflow due to the effect of gravity. In hanging the limb down, rubor appears due to pooling of blood in the capillaries and insufficient force at the arterial end of the loop to keep movement rapid. The rapidity

ency assured before an elective procedure is done on the foot even to the cutting of an ingrown toe nail.

Treatment.—

1. Care of the feet: General measures to prevent minor trauma to the feet will obviate many of the disasters that may occur. The well-known paint advertisement, "save

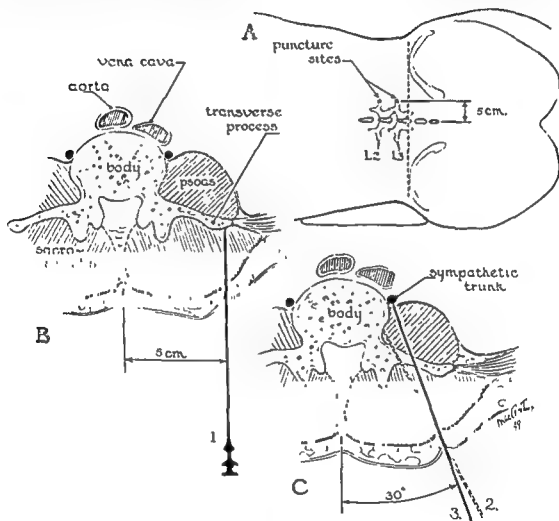


Fig 266.—Technique of lumbar block.

of the development of this rubor and its degree are also good indications of the degree of arterial ischemia. A foot which rapidly develops a deep rubor is one with a severe degree of arterial block. A small area of gangrene or infection may be the first signal of arterial disease. These usually result from trauma of some sort to the foot and point a moral that the arteries of every foot should be examined properly, and pat-

the surface and you save all," applies here. Daily washing is obligatory as is the wearing of soft wool socks. The feet should be oiled with lanolin or cold cream every second day and shoes should be pliable and loose fitting to prevent corns and blisters. Toe nails need careful cutting to prevent digging in at the edges, the best method being to cut the nails squarely and to scrape the convex surface of any nail that tends to

be ingrown Fungus infection between the toes should be energetically treated if it develops

2 Complete bed rest is indicated if gangrene or local infection is present Active

leg exercises should be carried out to prevent muscle atrophy where prolonged immobilization is necessary. Dry dressings only should be used on open areas, and antibiotics are useful where infection is present.

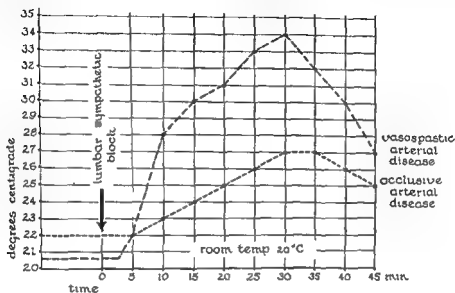


Fig 267—Typical skin temperature rises

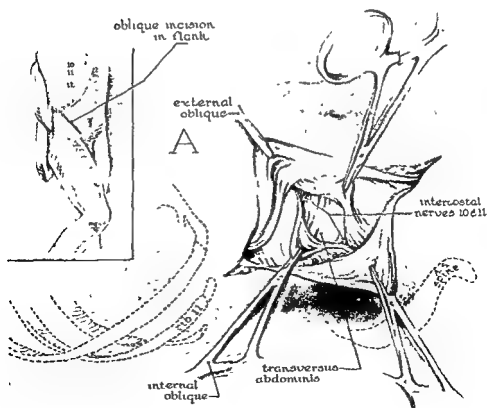


Fig 268—Technique of lumbar sympathectomy. (See continuation on next page)

3. Ethyl alcohol is a useful vasodilating agent and is also an aid in analgesia when pain is present. Liberal amounts should be used.

arm baths (110°-115° F.) or a baker to the abdominal area producing a state of general vasodilatation with benefit to the circulation of the legs.

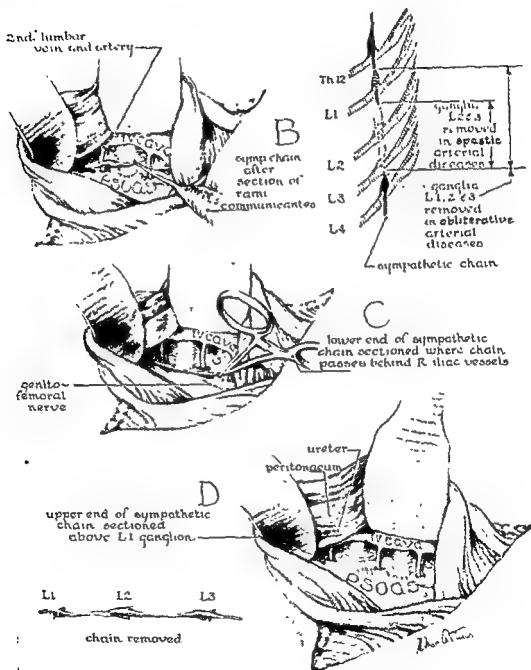


Fig 268.—Continued

4. Heat should not be applied to the affected part but can be used to good effect if employed to produce a state of general vasodilatation. This is accomplished by hot

5. Vasodilating drugs have not proved to be of much benefit because of their uncertain and transient effect. It is not reasonable that a badly damaged vascular bed suc

as occurs in advanced arteriosclerosis obliterans can show much vasodilatation. Also it must be realized that these drugs have a generalized action which will be major in the lesser involved vessels elsewhere in the body and so can possibly decrease the peripheral blood flow because of their action on the remainder of the body. Many supposed good results of these vasodilator drugs are really the result of the adaptive capacity of the vascular system. Over a period of months following the partial or complete occlusion of a main vessel, the functioning collaterals gradually dilate because of the greater volume of blood forced into them. Peripheral circulation improves and, if one of these drugs were coincidentally given, the improvement is credited to it. For example, ligation of the superficial femoral artery in a normal individual results in a loss of pedal pulses for only one to three months.

Many vasodilating drugs of variable usefulness are at present on the market. Their popularity is evanescent and it is beyond the scope of this book to discuss each separately. Many more will undoubtedly appear in the near future. Those currently in vogue are tetra-ethyl-ammonium chloride, nicotinic alcohol, Priscoline and diergocornine.

6 In arteriosclerosis the collateral vessels may still show potential vasodilatation. This can be determined if a rise in skin temperature of the foot follows procaine block of the lumbar sympathetic chain or the administration of tetra-ethyl-ammonium chloride. If such a true rise occurs of over 1°C following these tests, then maximal permanent vasodilatation can be accomplished by the operation of lumbar sympathectomy.

The patient should be warned, however, that this operation is not a cure, but only a method of improving to some degree the skin circulation to the legs. Claudication is frequently not improved but the feet feel warmer, ischemic neuritis is improved, and indolent infections are healed in the properly selected case.

7. Amputations are the eventual result in a relatively large number of cases of arterio-

sclerosis obliterans due to irreversible ischemia and gangrene. The majority of such amputations are done at the lower thigh level because of the poor distal circulation which mitigates against healing of the operative wound. At the Royal Victoria Hospital from 1936 to 1946, 141 major amputations were performed of which 101 were done because of the results of arterial disease. Eighty-five were done through the mid or lower third of the thigh, 13 were end-bearing operations at the level of the knee, and 3 were at the site of election. These figures should not indicate that minor amputations are not employed because healing will take place in many local amputations of toes where the circulation has been properly and previously assessed.

ACUTE ARTERIAL OCCLUSION

The two main causes of this sudden and frequently disastrous condition are arterial thrombosis and arterial embolism. The differentiation between them is often difficult, but such a differentiation should be made if possible because surgical removal of an impacted embolus is indicated in the early case of arterial embolism but less often in acute thrombosis. However, only certain sites of lodgment of emboli need surgery, the most common being the bifurcation of the common femoral artery. Embolism of the upper limb rarely needs surgical removal as the collateral circulation is adequate when the blockage is distal to the axillary artery.

Both acute arterial thrombosis and embolism are of sudden onset but the latter is the more acute. As a rule, acute pain is experienced in the limb with the rapid development of numbness and semiparesis depending on the degree of ischemia. When total ischemia is present, blanching and then a mottled cyanotic discoloration are the distinctive color changes. Occasionally the above findings occur in the absence of pain and consequently a delay, which may be fatal to the limb, occurs in diagnosis. If ischemia is allowed to continue untreated,

anesthesia develops in the distal part of the limb, gradually progressing centrally. The calf muscles become firm to the touch and tender, with loss of all contractile power, and the rigidity of the powerful ankle flexors gives a plantar flexion of the foot. The distal extremity is cold, and, on palpation, a zone of transition between warm viable tissues and cold ischemic ones can be found. Visual evidence of demarcation is also present in this area. Later frank gangrene of the ischemic parts develops.

the typical petechiae of this disease. Such a blockage therefore is not severe in effect except in the brain. More rarely, a portion of a mural thrombus can be dislodged from an arteriosclerotic plaque in the aorta or from a proximal aneurysm. Such an embolus will progress distally and become arrested at a site depending on the size of the embolus. Rapid narrowing in size of an artery occurs at the sites of major branches or bifurcation and, as can be seen in the illustration, emboli become lodged at such points.

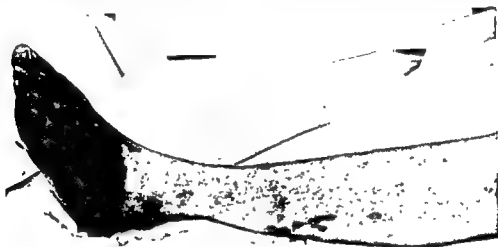


Fig 269—Gross leg gangrene, subsequent to neglected common femoral embolus.

The main points of differentiation between acute thrombosis and embolism are that embolism usually has a more sudden onset than thrombosis. The effect of an embolus lodgment is like a bolt out of the blue versus that of thrombosis which is somewhat more gradual and takes several hours to develop. In the case of embolism, there must be some focus from which an embolus could have originated. The focus is most commonly a mural thrombus in a fibrillating heart (left side of heart) or a similar thrombus which has formed following a recent cardiac infarction from coronary thrombosis. Again, a portion of valve vegetation may be dislodged from a case of bacterial endocarditis. However, in bacterial endocarditis the emboli are small in size and block lesser arteries and produce

Treatment.—The diagnosis of acute arterial occlusion is urgently necessary as is also the determination of the cause of occlusion so that therapy can be instituted before irreversible ischemic changes occur in the limb. The results of treatment vary directly with the length of time the occlusion exists and consequently it is imperative that the medical specialist and general practitioner who first see this case are impressed with the need for speed. After six hours of ischemia there is a sharp decline in the number of legs which can be saved and gangrene and subsequent amputation is the rule. As previously emphasized, the differentiation of an embolus from acute thrombosis is most important when the question of surgery arises regarding removal of the occluding thrombus. Surgery at the suitable time fre-

quently gives brilliant results in embolism but is less indicated in acute thrombosis

When first seen, and, particularly if less than six hours from onset, conservative therapy is indicated in both conditions. Priscoline is given in a 50 mg dose intravenously, the patient is transported to hospital, and anticoagulant therapy is begun. The leg is kept cool, and procaine block of the appropriate sympathetic chain is carried out to release the spasm of the remainder of the main vessel and the collaterals which usually occurs as a result of the acute blockage. If signs of improvement in the distal circulation of the limb do not occur within an hour of this therapy, then embolectomy is indicated in those cases where this diagnosis is beyond question or where reasonable certainty exists that such a diagnosis is cor-

sites of bifurcation of the main arteries should be kept in mind, as an embolus is most likely to plug an artery where an abrupt change in its lumen results from the sudden giving off of a large branch.

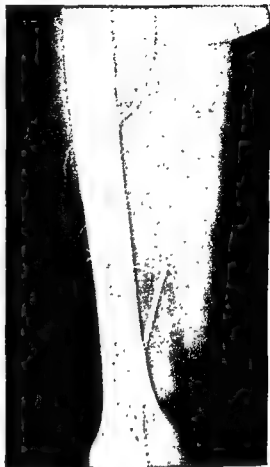


Fig 271—Arteriogram showing thrombotic occlusion of the proximal superficial femoral artery and collateral circulation around the area of blockage

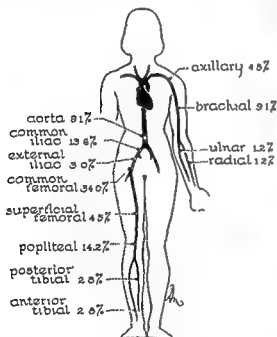


Fig 270—Frequency of embolus lodgment

rect. The diagnosis of embolism must also include a correct level of lodgment of the embolus. Such an assessment is made by digital palpation of the vessel pulsations, the site of pain and paresthesia, the determination of where the level of ischemia commences, and the use of the oscillometer. The

It is frequently difficult clinically to distinguish between acute embolism and thrombosis. An arteriogram can be readily performed and will be of great benefit in giving the correct diagnosis. In a dubious case, if conservative therapy has not shown improvement in the distal circulation, then surgery should be attempted in an effort to save the foot. If a thrombus is found occluding a major artery, as for example, the superficial femoral, the surgeon should be

prepared to incise the full length of the vessel in the thigh to extract the clot. The artery is then repaired and general or regional heparinization commenced to prevent reformation of the clot at the suture line. Regional heparinization is best accomplished by the insertion of a small polyethylene tube into the artery just proximal to the suture line. A constant slow heparin drip is carried out through this tube with sufficient heparin to prevent local clotting but not enough to disturb, when diffused, the general clotting mechanism.

Certain sites of lodgment of emboli or sites of acute thrombosis do not require surgery and will respond well to conservative therapy. The anterior and posterior tibial arteries when blocked will not result in severe ischemia, and the same can be said for the radial and ulnar arteries. Popliteal and brachial embolectomy are also seldom required.

ARTERIAL CONDITIONS SUBSEQUENT TO AUTONOMIC IMBALANCE

Raynaud's Disease and Raynaud's Phenomenon

Under this heading is included a group of closely allied conditions, the result of arteriolar spasm rather than organic occlusion. All the varieties resemble one another but in different degrees. Classical Raynaud's disease consists of attacks of arteriolar spasm most commonly affecting the hands and rarely the feet. Women preponderate in the ratio of 4 or 5:1. Cold is a precipitating factor in the majority, but emotional stress may also bring on attacks indicating the fact that the emotionally unstable individual is more prone to this disease than the stable, well-adjusted person. True Raynaud's is differentiated from Raynaud's phenomenon by the fact that the attacks are clear cut, relatively abrupt in origin, and consist of a period of intense vasoconstriction lasting 5 to 20 minutes when the fingers are white

and numb, followed by deep cyanosis and finally a burning erythema of the involved digits. In Raynaud's phenomenon, the same etiology is present but the effects are slightly different. Blanching is not so dramatic and the return to normal color is gradual and does not show the stage of cyanosis or erythema. There is the occasional case where the etiology appears to be due to the use of vibrating tools such as the pneumatic hammer.

Diagnosis.—The history in such a case is the most important point in establishing a diagnosis. Attacks of blanching of a finger or fingers gradually increasing in severity and possibly gradually involving more and more of the hand are characteristic. The presence of the "three color phase" in such a case is pathognomonic of Raynaud's disease. The preponderance in women, involvement of both upper extremities, relatively early onset (aged 20 to 40) and the precipitating factors of cold and emotional tension should give a clear diagnosis in the absence of any signs at the time of examination. During an attack (which frequently can be precipitated at will by exposure to cold), varying degrees of blanching are seen from dead white to a yellowish hue. The fingers feel numb and clumsy. The radial pulses are normal as far as palpable pulsations are concerned because the condition is arteriolar in type. Raynaud's attacks can be initiated by the conditions which produce neurovascular compression in the neck such as cervical rib and scalenus anticus syndrome. Such a cause should be ruled out before a diagnosis of idiopathic Raynaud's disease is made.

In the occasional severe case with frequently repeated severe episodes of spasm, organic changes appear in the digital arteries. These consist of thickening of the arterial walls and occasionally thrombosis. In such a case, it is not uncommon for infections to occur about the finger tips, chiefly about the nails. These are chronic and

painful and may result in local patches of superficial gangrene.

Scleroderma of the fingers and hand is occasionally seen as a complication to Raynaud's disease. The skin becomes atrophied and tightly stretched and the deeper tissues are fibrosed even to the joints. Circulation is further decreased by this process and organic vascular occlusion occurs with resultant trophic changes in the digits. Gangrene and chronic ulceration frequently supervene.

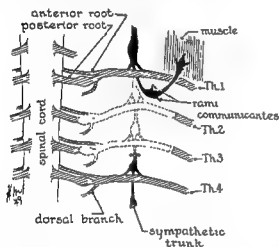


Fig 272—Diagrammatic illustration of technique of preganglionic upper dorsal sympathectomy

Treatment.—It must be kept in mind that the great majority of cases of Raynaud's disease and Raynaud's phenomenon are mild and never seek medical advice. They have discovered for themselves that if they protect their hands from cold, wear warm clothing in winter and avoid handling cold objects, they will be relatively comfortable. The removal to a warm climate in the winter may be necessary. Emotional states are harder to control, but reassurance and careful medical guidance are of benefit. In the severer cases and especially where organic changes are occurring in the finger tips, surgical treatment is indicated. Preganglionic section of the sympathetic fibers to the arm gives spectacular initial results.

According to Smithwick's technique, the upper dorsal sympathetic chain is sectioned

below the third thoracic ganglion and the white and grey rami to T 2 and 3 are sectioned, the former being removed intraspinally by intraspinal section of the somatic nerves T 2 and 3. The proximal portion of the sympathetic chain is then removed as far as possible from the field by suture of its free distal end to the muscles of the back.

Results from this operation unfortunately are not permanent, and about 60 to 70% of cases tend to recur to some degree after six months. The reason for this recurrence is not clear, local arterial sensitization to circulating adrenalin has been cited as a factor. Sensitization of the arterioles to cold or regeneration of the interrupted sympathetic pathways has also been blamed for recurrence.

More recently a return to the older surgical technique of removal of the stellate, first and second dorsal ganglia has become popular. It has been shown that better long-term sympathetic denervation of the upper limb can be achieved by this method. The only disadvantage to this operation is the production of a Horner's syndrome.

VASOMOTOR CHANGES RESULTING FROM TRAUMA AND INFECTIONS

When tissue is damaged by trauma or an infective process, the resultant healing is dependent to some extent on a normal nervous control of the reparative process. In the occasional rare case there appears to be an abnormal nervous control which may show a preponderance of the vasoconstrictive (sympathetic) tone or rarely vasodilator preponderance. The degree of such abnormality varies greatly, and the lesser degrees are transient and pass off before serious effects are noticed. However, in the occasional case, a chronic state of either vasoconstriction or vasodilatation of the vascular bed results. Such a continued effect leads to an abnormal process which has a widespread result in the limb and shows, in the spastic type, clinical evidence of vasoconstriction and coolness of the limb, in-

creased sweating, pain, and vague, poorly defined sensory changes in the limb. The peripheral pulses appear to be reduced, edema may be present, and the joints become stiff and painful. The vasodilatation type shows a limb which is warm, painful, dry, edematous, and which later may show a typical spotty decalcification of bone known as Sudeck's atrophy. The pain in both types is typically causalgic in nature and a case may begin by showing the vasodilatation phase which later (2 to 3 months) changes over to the vasoconstriction type.

Trauma of a mild degree, such as that resulting in a sprain, may be the initiating factor and the author has seen cases resulting from burns, minor fractures and even minor surgical wounds such as an incision for drainage of an abscess. Infections in the limb such as cellulitis or phlebitis may be the originating cause. This whole condition is somewhat vague and poorly understood and the exact nerve pathways taken by these abnormal reflexes is not clearly known. Why one person will be affected and not another from identical trauma is not known, but it appears as though this condition is more likely in those persons who show instability of the nervous system, especially the autonomic portion.

Treatment.—The fact that a neurovascular imbalance is present is utilized in treatment, and interruption of some portion of the reflex arc is logical therapy. The sympathetic supply to the limb is the efferent arc to this reflex and it is simple to interrupt. Procaine block of the appropriate sympathetic chain will often give dramatic temporary relief of symptoms which lasts for a progressively increasing length of time after each block and, after the second or third, the curative effect may be permanent. If a temporary effect only is noted after each block, then a sympathectomy will give a permanent effect. In the later cases where osteoporosis is severe, edema has been present for a long time and joint stiffness is present, long-continued physiotherapy is a necessity in producing recovery.

DISEASES OF VEINS

Varicose Veins

A varicose vein may be defined as an abnormally dilated vein, and, for practical purposes, the legs and lower abdomen are almost solely involved. These veins show an increase in caliber, sacculations due to patchy thinning of segments of the vein wall, elongation and tortuosity and fibrous thickening of other portions of the wall. A loss of valvular function takes place, and at times calcification of the vein wall may occur.

Etiology.—There are two main types of varicose veins from the causative standpoint. The *primary* ones develop in early adult life and probably arise on the basis of a congenital defect in the structure of the vein. This structural defect is often hereditary, as a tendency to varicose veins is often seen in certain families. Such a defect is likely a weakness of the wall and valves of the veins. Also a decreased initial number of valves may be present, the normal number being 6 to 8 in a competently functioning great saphenous vein. This congenital and hereditary background is probably the reason that pregnancy will cause the appearance of varicose veins in one woman and not in another, or why one shopgirl who stands all day will develop varicose veins whereas another will not.

Secondary varicose veins are those that develop following increased venous pressure acting upon them from the main venous trunks. These follow a previous deep phlebitis where increased venous pressure can occur in the damaged deep vein from direct transmission of increased intra-abdominal pressure. This increased pressure is transmitted to the superficial veins and structural defects in the valves and vein walls occur with the development of true varicose veins. Another example of this mechanism is the development of hemorrhoids and esophageal varices in portal hypertension.

Signs and Symptoms.—In some cases, well-developed varicose veins result in no

symptoms whatsoever and patients present themselves for treatment from the cosmetic standpoint. However, the usual complaints are those of tiredness, heaviness, fullness and aching of the leg after prolonged standing and therefore worse toward the close of the day. Menstruation usually aggravates the complaints. Symptoms of uncomplicated varicose veins should be completely relieved by recumbency with the possible exception



Fig. 273—Extensive primary varicose veins involving both legs

of nocturnal muscle cramps. To give the above symptoms, the veins should be well developed and should show a positive Trendelenburg test (a reversal of flow in the vein when the patient assumes the upright position). Beware of falling into the common error of blaming leg symptoms on a few varicose veins especially where the symptom complex is at variance with that above mentioned!

An essential part of the investigation of a case of varicose veins is the proper assessment of the degree of incompetency of the affected veins and valves and a determination of the level the incompetence occurs. Upon this knowledge the entire question of treatment depends. A number of tests have been designed to answer this point and all are modifications of the original Brodie-Trendelenburg test. This test is done by having the patient horizontal and lifting the affected leg. The blood drains out of the superficial veins assisted by massage centrally with the hand. With the veins collapsed, a rubber tourniquet is placed around the upper thigh sufficiently taut to compress the superficial veins only. The patient stands with the tourniquet on and the veins below the tourniquet should remain collapsed up to 30 seconds if the communicating veins and small saphenous vein distal to the tourniquet are competent. Release of the tourniquet will now give rapid filling of the distal varices from above when incompetence of the valves of the saphenous vein only is present. If, with the tourniquet on the thigh and the patient standing, the distal varices fill immediately, this indicates an incompetent valvular mechanism between the deep and superficial venous systems below the level of the tourniquet and the small saphenous vein can be included in this group of communicating veins. The site of the incompetent communicating vein or veins can be determined by repetition of the test using multiple tourniquets placed at different levels or by moving the single tourniquet gradually more distal until a level is found where the distal varices do not immediately fill but only fill when the tourniquet is released. At this level one is therefore just below the point of the most distal incompetent communicating vein.

Treatment.—

(A) *Primary Veins*—Where small dilated veins are present with no symptoms and the patient shows no reverse flow in the veins on performing the Brodie-Trendelenburg

test, then local injections of a sclerosing fluid are indicated if the patient wishes treatment for cosmetic reasons. When reverse flow occurs in the great saphenous vein alone and there are no incompetent communicating veins, a choice of surgical procedures can

be employed. Ligation of the great saphenous vein as close to the femoral junction as possible will interrupt the incompetent vein at its source. The vein can then be obliterated by the injection of sclerosing solutions either at time of operation or subsequently.

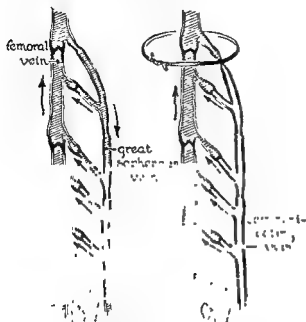
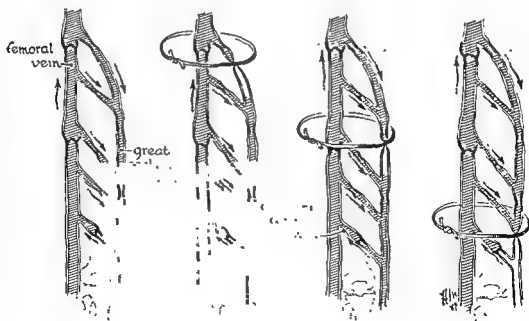


Fig 274—Diagrammatic steps in the performance of a Trendelenburg test.

valves of great saphenous vein incompetent ~
valves of communicating veins competent ~



valves of great saphenous vein incompetent
valves of several communicating veins incompetent

These solutions must be used with care because of several possible complications. Allergic manifestations are not too rare especially in those persons who have an allergic

phylactic shock. Another danger in the use of sclerosing fluids is the fact that when injected into a superficial vein a certain proportion of the fluid inevitably gets into the

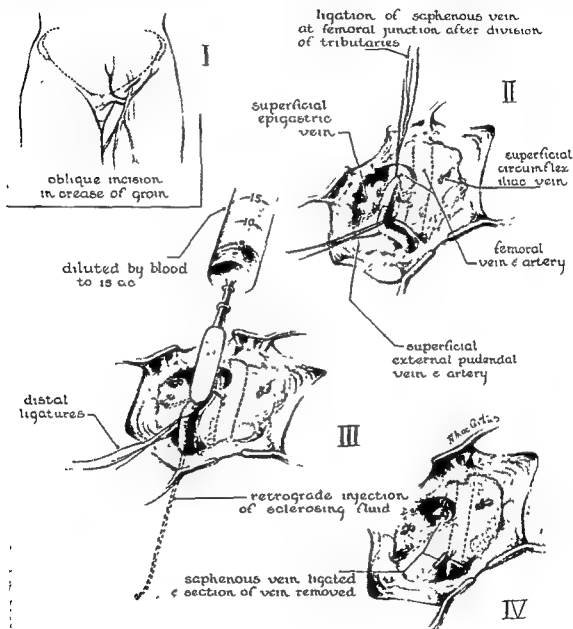


Fig 275A—Steps in the technique of saphenous vein ligation, and retrograde injection

tendency. Preoperative testing for sensitivity should be done in all cases before injection. Neglect of this precaution has resulted in many cases of sudden death from ana-

deep veins. This may occasionally result in deep venous thrombosis with crippling damage to the leg and the occasional case of pulmonary embolism.

Stripping of the great saphenous vein is probably a better procedure in all cases of incompetent varicose veins, as removal of the diseased vein results in a reduced tendency to recurrence. Stripping should be the only surgical procedure in those cases which show evidence of incompetent communicating veins because of the high rate of recurrence in cases when the ligation and injection technique is employed.

In about 20% of cases the small saphenous vein shows incompetent valves, and this vein will also need surgical attention. It should be ligated as high in the popliteal fossa as possible, and then distally injected or stripped according to the preference of the surgeon.

cases, but the degree of the recurrence will depend to a great extent on the excellence of the job done by the surgeon. Do not guarantee to the patient that permanent cure will result from the operation. The dentist does not guarantee after he has repaired one tooth that another will not break down.

(B) *Secondary Veins*—When varicose veins occur as a result of some other vascular condition, the treatment may vary according to the type of original disease. For instance, those following a congenital arteriovenous fistula will be only temporarily improved by the usual methods of vein treatment because the initial lesion remains



Fig 275B—Vein stripping, using Babcock stripper. Removal of complete vein from ankle incision

Varicose veins are not diseased tissue that can be obliterated or removed with any degree of permanency. The student should realize that he is dealing with a gradually progressive condition involving all the superficial (and possibly to some extent the deep) veins of the legs and that removal of one set of veins will not prevent enlargement of remaining ones. Some evidence of new varicose veins following surgery occurs in most

as before. Varicose veins following a deep phlebitis have been previously neglected under the assumption that these veins are part of the collateral return flow following damage to the deep circulation. However, if such varicose veins show reverse flow on standing, and therefore a positive Trendelenburg test, then these damaged veins only aggravate the venous stasis and should be corrected.

The method of correction should be by the stripping technique as the use of sclerosing fluids is dangerous due to the already damaged deep circulation. The patient should be warned prior to the operation that this procedure is not totally curative but will improve to some degree the venous stasis in the damaged leg. The instructions necessary for the care of the postphlebotic leg must still be carried out because of the continuing deep vein damage. The use of the elastic stocking as outlined will in many cases obviate the need for the surgical correction of these secondary varicose veins.



Fig 276—Typical varicose ulcer

Complications of Varicose Veins.—Venous stasis, whether from incompetent superficial varicose veins or from deep veins following a previous phlebitis, results in the occurrence of several abnormal physiological states. Increased venous pressures are present in the upright position and particularly after straining, with elevations up to 400 mm of water

from a normal of 50 to 100. This allows stasis of the capillary loop resulting in cyanosis, anoxemia of the endothelium allowing increased permeability and edema. This edema together with some decrease in O_2 and increased CO_2 tensions, also occasionally increased N P N. values, results in tissue devitalization and paves the way for the complications.

Varicose Ulcer.—When ulceration is present in association with varicose veins, these varicosities must be of a relatively severe degree and show valvular incompetence with reversal of flow on standing, otherwise such an ulcer is not a true varicose one. Varicose ulceration is usually preceded by the preliminary changes of chronic venous stasis. This results in brownish skin pigmentation, the result of the deposition of hemosiderin subsequent to the breakdown of extravasated red cells, also thinning of the skin with fibrosis and devitalization of the underlying fatty tissue. Chronic edema is frequently present aiding in the development of these changes and encouraging the presence of chronic low-grade periphlebotic infection and consequent induration.

With such devitalizing influences present, it is not uncommon, spontaneously or following a slight trauma, that an ulcer develops which persists as long as the chronic venous stasis is allowed to remain. This ulcer is most commonly located just above the medial malleolus, but may be anywhere in the lower half of the leg if trauma has been the initiating cause. Each time the ulcer recurs following inadequate treatment, further fibrosis is added to the former ulcer area rendering the tissues more devitalized and so more liable to further ulceration.

Treatment consists of correcting the underlying cause of the venous stasis by removal of the varicose veins. The ulcer is treated by some type of compression bandage to remove edema. The best dressing is still the Unna's paste boot. Bed rest, hot dressings, and the use of antibiotic drugs will be necessary in the acutely inflamed ulcers.

Varicose Eczema.—Another of the manifestations of chronic venous stasis is that of eczema. This usually consists of a patchy involvement of the medial ankle region but may be severe enough to involve most of the lower leg. It is manifest by a red, exuding, scaly involvement of the skin, and the patient complains of edema, burning and itching of the affected part. Again, it should be pointed out that, to be true varicose eczema, incompetent varicose veins must be present. A previous deep phlebitis, diabetes, or sensitivity reaction should be ruled out. Treatment consists of support to the leg, the same as for varicose ulceration, plus the correction of the incompetent varicose veins.

Superficial Phlebitis.—Varicose veins are more prone to the development of phlebitis than normally functioning ones because of the relative venous stasis present in addition to the fact that their exposed position renders them more liable to trauma. Such a phlebitis is diagnosed by the signs and symptoms of pain, redness, soreness and hardness in varicosities previously soft and painless.

Minor degrees of this complication are readily controlled by a pressure bandage such as Unna's paste boot or elastic adhesive dressing. There is no need for the patient to be immobilized. Major degrees involving a large stretch of vein, especially where extension toward the saphenofemoral junction is taking place, are best treated by high saphenous ligation at the saphenofemoral junction. This has the merit of preventing the rare case where the saphenous thrombus extends into the femoral vein with therefore possible embolus formation; also it corrects the basic fault in the original varicose veins. The inflammatory thrombus present obviates the need for the use of sclerosing fluid, and the same ultimate effects of obliteration of the varices take place. This method of therapy allows the patient to be ambulatory, corrects the underlying venous pathology, and so prevents recurrent attacks. A pressure dressing is applied

to the leg after operation to reduce the pain, stiffness and edema resulting from the phlebitis.

Venous Thrombosis

Thrombosis of blood in the venous tree is a complication fraught with danger to the patient's life and may result in varying degrees of mild to severe disablement. There are three principal factors which result in venous clotting:

1. Increased coagulability of the blood which may follow trauma or be secondary to anemia or polycythemia or may be present for some undetermined reason.
2. Slowing of venous blood flow which may occur secondary to heart disease, dehydration or prolonged immobility.
3. Damage to the intima of the vein resulting from trauma or infection.

The degree, extent, and type of the venous thrombosis vary according to the degree of associated inflammation, and may vary from the bland, quiet phlebothrombosis to the severe septic thrombophlebitis.

Bland Thrombosis (Phlebothrombosis)

Where the inflammatory element is mild and the intimal damage is minimal, blood clotting in the vein will take the form of a soft, loosely attached clot which gradually propagates centrally. Such a process most frequently originates in the veins of the lower extremities because in these veins the above-mentioned factors predisposing to thrombosis are most frequently met. Those of the sole of the foot, the calf and those of the adductor region of the thigh are most commonly originally involved. Propagation of the clot from these regions may take place into the main venous channels of the leg and progress centrally even as far as the vena cava.

The thrombus originates as a collection of platelets which adheres to the intimal endothelium at some area where intimal damage from whatever cause has taken place. Leucocytes and fibrin adhere usually in layers

The method of correction should be by the stripping technique as the use of sclerosing fluids is dangerous due to the already damaged deep circulation. The patient should be warned prior to the operation that this procedure is not totally curative but will improve to some degree the venous stasis in the damaged leg. The instructions necessary for the care of the postphlebotic leg must still be carried out because of the continuing deep vein damage. The use of the elastic stocking as outlined will in many cases obviate the need for the surgical correction of these secondary varicose veins.



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With such devitalizing influences present, it is not uncommon, spontaneously or following a slight trauma, that an ulcer develops which persists as long as the chronic venous stasis is allowed to remain. This ulcer is most commonly located just above the medial malleolus, but may be anywhere in the lower half of the leg if trauma has been the initiating cause. Each time the ulcer recurs following inadequate treatment, further fibrosis is added to the former ulcer area rendering the tissues more devitalized and so more liable to further ulceration.

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than formerly due to the improvement in surgical technique and the use of antibiotics. Certain medical diseases show a relatively high incidence of thrombophlebitis of the leg such as typhoid fever, pneumonia, Buerger's disease and ulcerative colitis. Occasionally thrombophlebitis is seen where no definite predisposing factor can be determined, but in such cases some infectious process such as an upper respiratory infection has usually preceded the onset.

Signs and Symptoms.—In the acute form, the patient will complain of a relatively sudden onset of a severe ache in the involved leg, variously localized in the calf, popliteal area, or thigh. The temperature climbs to 101° to 103°. Some cyanosis of the leg, even when horizontal, is usually present, and severe edema soon appears involving the lower leg only when the process starts distally or involving the whole leg and buttock when the femoral and iliac veins are affected. Acute tenderness is present over the involved area; the part is hot and full to the touch and pain results from muscular movement.

All gradations of thrombophlebitis occur and the lesser present the same picture but to a milder degree. Luke's sign also applies to thrombophlebitis and is an early diagnostic indication. A fulminating variety of thrombophlebitis is occasionally seen where the degree of involvement is sudden and severe and is associated with a major extent of arterial vasospasm to such a degree that the picture of acute arterial thrombosis or arterial embolism may be simulated. However, the leg edema and cyanosis will usually indicate the true picture. The arterial spasm may be of such a degree that true gangrene develops and such a lesion demands energetic treatment.

Pulmonary Embolism

As previously indicated, the origin of most pulmonary emboli is from the leg or rarely the pelvic veins and is usually the sequel to a case of bland thrombosis because of the

looseness of the attachment of the clot consequent to the minimal inflammatory involvement of the vein wall. In thrombophlebitis where inflammation of the vein wall is extensive, embolus production is uncommon (5%). Detachment of a fragment of clot occurs where a large tributary connects with the blocked main vein, the flow of blood from this tributary being sufficient to break off the "tail" thrombus. Sudden increases in intra-abdominal and consequently intravenous pressure may also produce the same result; this is the reason for the sudden death of patients in the act of using the bedpan or getting up for the first time.

The degree of pulmonary artery blockage depends directly on the size of the embolus, and the size of the embolus depends on the caliber of the vein from which it originates. A piece of clot from the veins of the lower leg will give a minor degree of pulmonary infarction while one from the common femoral or iliac may produce sudden death. Another factor which contributes to the degree of symptoms in pulmonary embolism is spasm of the bronchial tree and pulmonary vessels incident to the sudden lodgment of an embolus, this being similar to the effect of a peripheral arterial embolus. Another similar result is the fact that thrombosis can progress distally from the site of occlusion in the pulmonary artery and give a fatal termination to a patient who survived the initial catastrophe.

Signs and Symptoms.—Any person suffering from any inflammatory disease, after an operation or childbirth or who has been confined to bed for any period of time and who complains of a sudden pain in the chest should be suspected of having pulmonary embolism. Minor emboli may be manifested only by a "stitch in the chest" and be so mild that the initial one is not recognized. Pathognomonic is the triad of a sudden pain in the chest aggravated by respiration, breathlessness, and later hemoptysis. The greater the amount of lung infarcted the more marked are the symptoms. Pallor, cyanosis,

and a white thrombus is formed. Further clotting is mixed with red cells and a red thrombus is formed which progresses centrally with little or no attachment to the vein wall. Such a loosely adherent or freely floating clot attached only distally is known as a "tail" thrombus and consequently is easily detached to act as an embolus. Such a fragment finds lodgment in the lung to produce the typical pulmonary embolus and the amount of pulmonary artery occluded will depend directly on the size of the thrombus detached, those from a small distal vein producing minor emboli and those from a major vein may block the origin of the pulmonary artery producing sudden death.

Bland thrombosis may originally be associated with only a mild degree of inflammation of the vein wall but such an inflammatory involvement tends to increase in some cases and consequently an original case of phlebothrombosis may progress to a full-blown thrombophlebitis.

Signs and Symptoms.—It must be emphasized that this condition can occur both in medical and surgical diseases but probably is more common after surgery and childbirth. Symptoms may be completely absent, the first indication of the lesion being that of a pulmonary embolus. Many of these emboli are massive and produce the tragic occurrence of sudden death during an apparently uncomplicated convalescence. Signs and symptoms, however, usually occur first in the leg where the majority (95%) of such thromboses originate. Some aching in the foot, the calf, popliteal region or thigh will be noticed by the patient, and the leg will have the sensation of fullness and stiffness. Such symptoms usually occur or are aggravated when in the upright position (Luke's sign). Mild fever is present and the pulse is usually slightly elevated. Tenderness in the sole of the foot, the calf, popliteal region or medial thigh may be found and slightly increased warmth of these parts is usually present. A sense of fullness of the muscle mass of the calf is a very constant finding

A feeling of tightness or pain in the calf is produced in the majority of cases by forced dorsiflexion of the foot with the knee extended (Homan's sign). Mild edema of the ankle and lower leg is present and the foot may show some cyanosis on dependency. The sedimentation rate is elevated.

It cannot be emphasized too often that the condition of bland thrombosis and that of thrombophlebitis are part of the same process and differ only in the degrees of inflammatory involvement. Bland thrombosis has been described as an entity chiefly because of its minimal signs and symptoms and its great tendency to embolize (30% of cases). Consequently, with the possibility of such a serious complication, every effort should be made to prevent this development or to recognize it in its early stages. The condition should be constantly kept in mind and looked for in every postoperative patient, and a serious view taken of even the mildest leg complaint in any patient so that early therapy can be instituted and a possible pulmonary embolus averted.

Thrombophlebitis

As mentioned above, thrombophlebitis presents the picture of venous clotting associated with marked inflammatory involvement of the vein and to some degree, the perivenous structures. The same etiological factors pertain as in bland thrombosis, consequently it may occur following childbirth or a surgical operation, especially where infection is associated. The incidence is about 2% following surgical operations and is highest in kidney and bladder cases, major gynecology or such abdominal lesions as a perforated appendix. As in bland thrombosis, it occurs between the 5th to 15th postoperative or postpartum day and takes place in the leg veins in the great majority of cases though primary involvement of the pelvic veins may occur. The veins of the portal system occasionally are affected from some suppurative lesion in the abdomen but this condition appears to be much less common.

possible to say that the thrombus has not originated in the pelvic veins.

At the present time it is the author's opinion that there are only two indications for ligation in venous thrombosis. The first

taken. In this case, femoral ligations are usually inadequate, and ligation of the vena cava below the renal veins is the site of choice. The second indication is the ligation of the superficial femoral vein just dis-

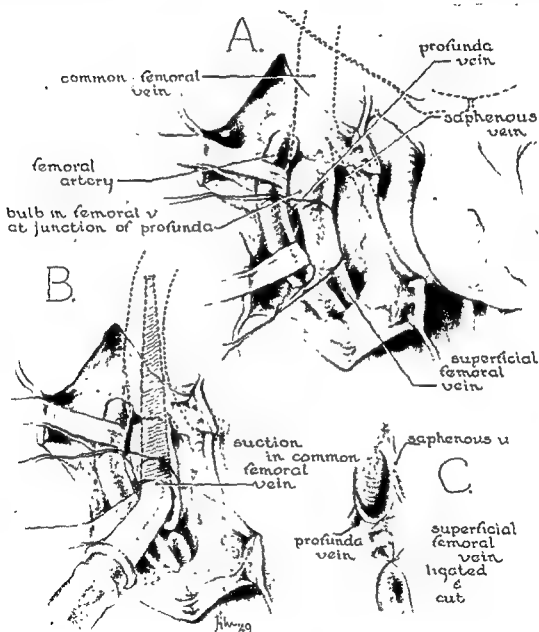


Fig 277.—Surgical technique for removal of thrombus in the common femoral vein.

is where the patient continues to throw multiple emboli to the lungs despite treatment by adequate anticoagulant therapy. His life is endangered and further steps must be

taken. In this case, femoral ligations are usually inadequate, and ligation of the vena cava below the renal veins is the site of choice. The second indication is the ligation of the superficial femoral vein just dis-

collapse, sweating, loss of consciousness, lowered blood pressure and rapid thready pulse are the signs of a major embolus. Differentiation of this picture from that of an acute coronary occlusion may be difficult, and many patients have died of so-called acute heart attack when the real reason was an embolus. The discovery of the site of the peripheral thrombus, the later spitting of blood, the development of a friction rub, and the x-ray appearance of a dense area in one lung will give the differentiation.

Treatment of Venous Thrombosis and Pulmonary Embolism.—Two relatively recent methods have been added in the treatment of this condition with marked improvement in the morbidity and mortality results. These are the employment of the anticoagulant drugs and the judicious use of vein ligation. The aim of present-day treatment is to make a diagnosis at the earliest possible moment and then institute active therapy to prevent extension of the process centrally. It is the emboli from the larger veins which kill or, in thrombophlebitis, it is the involvement of the major veins such as the common femoral or iliacs that produce the crippling sequelae in subsequent years of chronic swelling of the leg, eczema, and ulceration.

However, the best method of treatment is prophylaxis, and such prevention of this complication can be attained in great measure by careful attention to detail in the patient before and after operation. Anemia, dehydration, and alterations in the albumin-globulin ratio should be corrected prior to operation. Gentleness during the operation reduces the amount of tissue damage and consequently the amount of thrombokinasase liberated. Active movements of the legs should be carried out many times daily according to a definite schedule, and turning from side to side should be done hourly. Fowler's position should not be used, and tight binders or dressings on the abdomen should be discarded because they limit proper respiratory movements. The patient

should be out of bed as early as possible and encouraged to walk and not just sit in a chair. In a case of an elderly patient where extensive surgery has been done or where an increased clotting tendency is suspected by a history of previous episodes of thrombosis, anticoagulant drugs should be used as a prophylactic measure. The legs should be inspected and palpated daily in every case of major abdominal surgery, and treatment instituted at the first suspicion of venous thrombosis. Prophylactic ligation of both superficial femoral veins just distal to the profunda branch has been proposed by some groups prior to major abdominal surgery but the results achieved have not been impressive and do not justify the additional surgery.

The use of suitable vein ligation in the treatment of phlebothrombosis and thrombophlebitis has recently been through a phase of popularity but is now on the wane. The rationale of this procedure was to trap the thrombus distally by a suitably placed proximal vein ligation or if thrombus was found in the vein when the superficial or common femoral vein was opened then to suck out the central extension of the thrombus. The ideal site for such a vein exploration and ligation is the superficial femoral just distal to the profunda branch. Ligation at this level does not lead to leg edema or postphlebitic leg changes and the common femoral up to the common iliac can be cleared by a suction tip.

This reasoning is well in theory but is practically not so sound. In many occasions the author has found on opening the superficial or common femoral veins that clot is present which cannot be removed because of its internal adherence. In such a case clot reformation is inevitable. Other cases have occurred where a thrombus has formed central to the site of ligation, and resulted in embolism. Again it is frequently impossible to tell from which leg an embolus has originated, consequently a bilateral operation is necessary. Or again it is im-



Fig 278.—Retrograde venograms of the femoral system showing valvular destruction, partial recanalization and collateral dilatation following previous deep thrombophlebitis



Fig 279 —Postphlebotic ulcer with surrounding eczema and tissue fibrosis

bolization but to prevent the central extension of the process into the common femoral and iliac veins by dividing the extending pathway. If the process is kept distal to the major veins, convalescence is shortened but most important, the sequelae of the postphlebotic leg are prevented. It should be emphasized that all cases of deep venous thrombosis treated surgically should have coincident anticoagulant therapy.

Anticoagulant drugs will not dissolve an existing thrombus, but, if correctly used, should prevent its extension. If immediate anticoagulant effect is desired, heparin is used by the intravenous drip method or by intermittent intravenous or intramuscular injection. Dicumarol (200-300 mg) is started at the same time but, as its effect is not manifest until 36 to 48 hours, the heparin should be used for this period of time. The great majority of cases of venous thrombosis are at present treated by anticoagulant therapy and the below-mentioned adjuvant measures.

Where severe pain and edema are present in thrombophlebitis, it is an indication of more than usual arterial and venous spasm. In such cases, blockage of the appropriate lumbar sympathetic chain will relieve this vasospasm and aid recovery. One to four such procaine blocks lessen pain, reduce the edema and appear to shorten convalescence. Other adjuvant methods in the treatment of acute thrombophlebitis are elevation of the leg as high as is comfortable and active contraction of the muscles of the limb, the first to aid in draining off the edema and the second to aid also in this respect and to prevent muscle atrophy during convalescence.

When a minor embolus occurs, the treatment consists (as above outlined) in energetic attempts to prevent a second. However, a major embolus demands not only treatment from this aspect but also emergency therapy to prevent death as a result of the embolus because there is no doubt that immediate attention to the pulmonary effects of such an embolus will result in

fewer deaths. Many patients who die several hours after a pulmonary embolus could probably have been saved by intelligent treatment. When an embolus lodges in a major branch of the pulmonary artery, as when an embolus lodges in a peripheral artery, a certain degree of spasm of the arterial tree occurs and, in the lung, also bronchial spasm. The parallel is also present in respect to distal extension of thrombosis from the point of obstruction. Treatment therefore should be designed to combat these factors. Pure oxygen is given by B L B mask to counteract anoxia, papaverine in half grain doses intravenously will help relieve the arterial and bronchial spasm, and anticoagulants properly administered will prevent distal propagation of the thrombus.

Postphlebotic Leg

The ideals in the treatment of leg thrombophlebitis are twofold, namely, to terminate the disease process as quickly as possible, and also to lessen or eliminate the late sequelae of this disease. It is unfortunately too frequent that the condition is well advanced when energetic treatment, as above outlined, is initiated or again the result of treatment which has been only half hearted. When the thrombophlebotic process has reached and involved the common femoral and iliac veins the unfortunate individual is left with some degree of permanent damage to the leg. When the disease is confined distal to the bifurcation of the common femoral vein, then permanent postphlebotic changes in the leg are rare.

The inflammatory involvement of the vein results in thrombosis in the vein lumen and destruction of the vein valves. This thrombus later recanalizes in whole or in part in most instances, but the vein remains an inelastic valveless tube subject to increases in venous pressure on straining (transmitted retrogradely from the abdominal systemic veins). The inflammatory process also involves the perivenous structures and, in the common femoral and iliacs,

currence is almost inevitable unless care is taken to prevent edema and injury to this unhealthy leg.

Phlebitis Migrans

This variety of venous thrombosis is relatively uncommon and consists of multiple waves of thrombophlebitis in short segments of superficial veins of the limbs and occasionally the trunk. This thrombophlebitis occurs in normal veins as distinct from that occurring in varicose veins. The patient complains of hard, reddened tender subcutaneous cords in the location of the superficial veins. One vein segment after another becomes involved and the condition may last for months. Such a lesion occurs in 30% of cases of thromboangiitis obliterans, and a careful examination of the arterial tree is indicated for many months to rule out this possibility. However, most cases of this condition do not show any immediate or late evidence of thromboangiitis and therefore constitute separate entities. The etiology is not clear, but the author has had many cases where a careful search for focal infections and their removal has resulted in cure. The tonsils are the most important focus and should be removed on the slightest suspicion. Anticoagulants are of little use because of the chronic nature of the disease.

VASCULAR LESIONS DUE TO COLD

I. Cold Sensitivity

A rare vascular phenomenon occurs in certain patients who show an intense reaction to cold. This takes the form of a severe urticarial reaction in the skin area affected by the cold. Swelling, pruritus, edema, and blebs occur. If a sufficient area is affected, general symptoms are present resembling histamine overdose and it appears as though the condition results from excessive histamine release by the cold stimulus. In major cold exposure, drop in blood pressure with syncope, nausea and vomiting occur, associated with localized urticaria. The diag-

nosis is suspected by the history and can be confirmed by the local application of cold to the skin which results in the typical urticarial reaction. Treatment consists in avoiding cold exposure and graduated injections of histamine to desensitize the individual.

II. Frostbite

The effects of exposure to cold are relatively uncommon at the present time, but in wartime it is a most important factor to be guarded against in troops operating in winter or Arctic climates and also in high altitude fliers. A large amount of research has been done in the field of prophylaxis during the recent war and also recently due to the possibility of an Arctic campaign.

Sustained cold operating on normal skin and deeper tissues causes primary vasoconstriction and stasis in the arteriole and capillary. This leads to "silting" of the blood in the vessel and thrombosis. Tissue cells become solidified and may rupture. On thawing, there is marked transudation of fluid from the damaged vessels due to changes in the permeability of the endothelium and this results in the typical swollen appearance of a frozen part. This swelling associated with the local tissue damage and the vascular thrombosis results in marked ischemia to the part and is the cause of the gangrene. Such gangrene is always more extensive on the surface, a point which should be kept in mind when amputation becomes necessary. Also it should be remembered that frostbite will occur more readily and in lesser degrees of cold in persons with already existing peripheral arterial disease.

The prophylaxis of frostbite is of major importance to the present-day Arctic explorer, in military winter campaigns or the high altitude flier. Properly heated flying clothes have been effective for the airman but the army still has too high an incidence of frostbite, because of prolonged standing in fox holes with damp feet or casualties lying unattended for long periods. The recent

this means involvement of the main lymphatic trunks from the leg which lie close to the vein in these areas. Variable degrees of lymphatic obstruction result from the perivenous scarring and the individual is left not only with inadequate venous return from the leg (particularly when in the upright position) but also abnormal lymphatic drainage.

This combination of permanent damage results in chronic edema of the leg after prolonged standing. This edema leads to chronic malnutrition of the tissues in the lower leg and consequent decreased resistance to infection and precipitates the complications of the postphlebotic leg. These complications may present themselves one to twenty years after the acute phlebitis, depending on the degree of initial damage of the original process and the extent to which the individual had to be upright in the interval. One of the complications most frequently encountered is that of chronic ulceration. This is seen most commonly just proximal to the medial malleolus. This ulceration should be easily differentiated from the true varicose ulcer by the history of previous deep phlebitis, the absence or paucity of varicose veins, and the other soft tissue changes of deep phlebitis. Other complications are those of eczema involving the lower leg (stasis dermatitis), areas of induration, pigmentation and subacute inflammation, and the development of secondary varicose veins.

Prevention of the complications of the postphlebotic leg should be the primary aim of treatment. The patient who has recovered from a severe thrombophlebitis should have his condition explained fully. It should be stressed that the leg will never be normal again and that complications can occur if the chronic edema is neglected. A strong elastic stocking should be worn for an indefinite period, employment entailing prolonged standing should be avoided, and the patient should get the habit of elevating the leg whenever possible. Once an ulcer has developed, bed rest with leg elevation and

moist hot compresses will result in healing but recurrence is likely in the future unless the following instructions are carried out.

Instructions for the Continual Care of the Leg Damaged by Phlebitis

1. Wear your elastic stocking from the time you get out of bed until you retire, with the exception of bath time. The stocking should be renewed every three months, and it is best to have two stockings that can be alternated for cleaning purposes.

2. Do not stand for more than thirty minutes without sitting down for fifteen minutes and elevating the leg on another chair. When standing get into the habit of flexing the toes in your shoes and frequently rising on tip toes.

3. Plan your day so that you can lie down for two to three half-hour periods and elevate your leg to a 45 degree angle. The back of a small straight-backed chair is useful for this purpose.

4. Whenever you sit down, elevate your leg on a foot-stool, chair or chesterfield.

5. At night raise the foot of the bed on blocks about six inches.

6. Apply a bland cold cream to the affected skin at night about every second day.

7. Avoid irritation to the involved leg, especially in respect to sunburn and hot water bottles.

■ Be extremely careful to prevent bumping, bruising, or scratching the affected leg.

In the lesser degrees of ulceration, ambulatory treatment with the Unna's paste boot or one of its modifications will result in healing, but, again, permanent care of the leg is necessary.

The occasional case of postphlebotic ulceration will be encountered where the ulcer is so large, fibrotic and long standing, that excision and skin grafting are necessary for initial healing. Also some degree of sympathetic dystrophy is seen occasionally in this type of case and a lumbar sympathectomy will speed ulcer healing and minimize the possibility of recurrence. As mentioned previously, the removal of secondary incompetent varicose veins in the postphlebotic leg will also accomplish the same result. But it cannot be too often emphasized that all treatment is merely partial, the underlying etiological pathology still remains, and re-

artery, the result of prolonged trauma. Or the picture may be that of occlusion of digital arteries to the fingers presumably on the

is present in the subclavian artery, the distal pulses will be impalpable or weak depending on the efficiency of the collateral circulation.

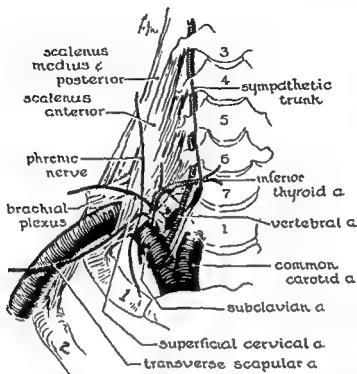


Fig 280.—Anatomy of the scalenus area.

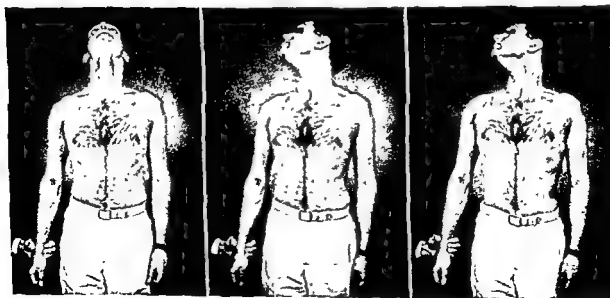


Fig 281.—Steps in the performance of the Adson maneuver.

basis of prolonged spasm. Cold bluish-red fingers result with pain and trophic changes which may be severe enough to produce digital gangrene. When arterial occlusion

The blood pressure in this arm will be low or unobtainable.

The patient will volunteer the information in many cases that carrying heavy objects

Korean campaign has emphasized the need for mass instruction in prophylaxis concerning the effects of cold.

Treatment consists in extreme gentleness to the frozen part both during and after the actual freezing. Rubbing or the application of snow is contraindicated because the frozen cell is easily damaged. This tissue should be thawed as rapidly as possible at a temperature just above body heat. The area is rendered antiseptic and enclosed in a sterile dressing to minimize bacterial invasion of the ischemic tissue. The use of heparin as soon as possible has been shown to lessen the vascular thrombosis and the consequent anoxia and later gangrene. Such agents as rutin to lessen capillary permeability and the antihistamine drugs are of dubious use. The latter is supposedly of use because it has been shown that there is an increased histamine content in frozen tissue. Sympathetic blocks are theoretically useful but have not proved too efficacious in the early case. When gangrene develops, it should be kept dry and treated conservatively for a long period until a definite line of demarcation appears. Frequently it will be found that only local amputations will be necessary when originally it was thought that a foot or hand might be lost. Sympathectomy in the later stages is useful in decreasing the sequelae of cold sensitivity, pain and vasospasm.

III. Trench and Immersion Foot

These two lesions are similar and are the result of cold and dampness or immersion in cold water over a prolonged period. Added factors are the wearing of tight shoes during this time or sitting in a lifeboat with the legs dependent for long periods. The temperature is not sufficient to produce actual freezing of tissue, but when operative over a period of many hours produces pathological changes somewhat similar to frostbite, especially in regard to vessel thrombosis, swelling and tissue anoxia. This anoxia affects all tissues, even bone, and the nerve damage which occurs may be one of

the most crippling of the late effects. When removed from the cold environment, marked edema, hyperemia and severe pain occur. Superficial gangrene may result.

Disability is severe and prolonged and treatment is similar to that of frostbite. The results of the ischemic neuritis persist for months resulting in varying degrees of paresthesias and pain. As in frostbite, the gangrene should be treated most conservatively.

COSTOBRACHIAL COMPRESSION SYNDROME

There is a group of anatomic or developmental anomalies of the shoulder girdle which by compression of the components of the brachial plexus and/or the subclavian artery can cause a variety of signs and symptoms involving the upper extremity. These conditions are cervical rib, scalenus anticus syndrome, costoclavicular syndrome, and abnormalities of the first thoracic rib.

Signs and Symptoms—These are common to all the above conditions and can be divided into two main groups, those with evidence of brachial plexus pressure and those with indications of interference with the vascular supply to the limb. A combination of both types may be present. The neurological picture consists of the symptom of pain localized to the deltoid area or down the arm most commonly in the distribution of the medial cord of the brachial plexus. The individual nerve most frequently affected is the ulnar because its origin from C8 and T1 is most subject to compression due to its anatomical position. The degree of involvement varies from paresthesias to partial motor and sensory loss. The vascular symptoms include attacks of arteriolar spasm resembling Raynaud's phenomenon, these attacks probably being caused by pressure irritation of the sympathetic outflow to the limb, this outflow running with the medial cord of the brachial plexus and with the subclavian artery. Evidence of organic arterial occlusion is sometimes present which may be severe due to thrombosis of the subclavian

artery, the result of prolonged trauma. Or the picture may be that of occlusion of digital arteries to the fingers presumably on the

is present in the subclavian artery, the distal pulses will be impalpable or weak depending on the efficiency of the collateral circulation.

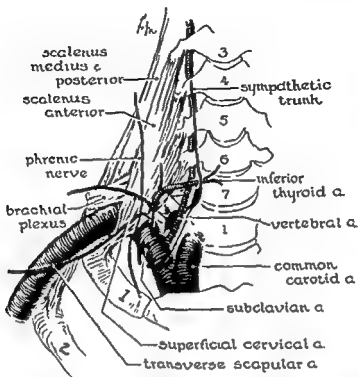


Fig 280—Anatomy of the scalenus area.

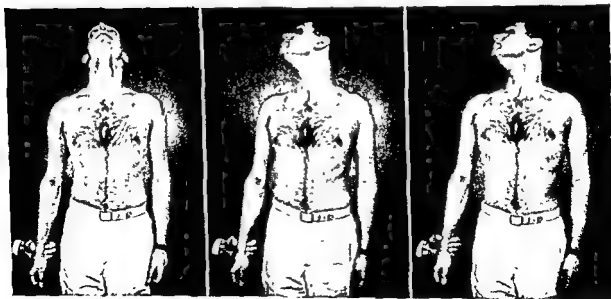


Fig 281.—Steps in the performance of the Adson maneuver.

basis of prolonged spasm. Cold bluish-red fingers result with pain and trophic changes which may be severe enough to produce digital gangrene. When arterial occlusion

The blood pressure in this arm will be low or unobtainable

The patient will volunteer the information in many cases that carrying heavy objects

in this hand will aggravate symptoms, also that he cannot sleep on this side at night because the hand and arm "go to sleep" and develop pins-and-needles sensations. He finds greater comfort by sleeping with his arms above his head. A definite test (Adson's maneuver) has been devised which indicates that subclavian artery compression is taking place. This is designed to indicate scalenus compression but applies equally

well to the other varieties of shoulder girdle compression. A perusal of the illustrations will indicate the performance of these tests.

In the differential diagnosis of these compression syndromes, especially where pain and neurological signs are present, certain other conditions should be considered and ruled out before a positive diagnosis of shoulder girdle compression can be made. Prolapse of a cervical intervertebral disc is a common cause of this so-called brachial neuritis as is also nerve pressure following osteoarthritis involving the intervertebral foramina. The medical conditions of neuritis subsequent to diabetes, alcohol, lead poisoning and vitamin B deficiency should also be kept in mind.

Cervical Rib

About 0.5% of all persons have a cervical rib arising from the 7th cervical vertebra. This rib is of all varieties from small stubs to a complete rib articulating with the first thoracic rib. Also seen is a short bony rib with a cartilaginous or fibrous cord articulating with the first rib in the region of the scalene tubercle. These accessory ribs are usually bilateral but frequently only one side gives symptoms. The medial cord of the brachial plexus, the sympathetic supply to the arm, and the subclavian artery have to

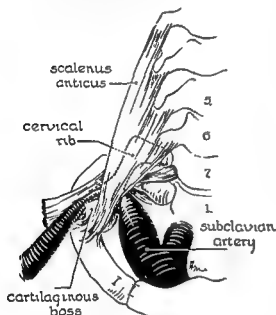


Fig 282.—Case of subclavian artery compression due to cartilaginous extension of cervical rib



Fig 283.—Bilateral cervical ribs, only that on the right gave symptoms.

pass over this rib before descending into the arm and consequently are subjected to pressure. However, many persons have cervical ribs without symptoms and in many others symptoms do not develop until middle life. The late development of symptoms in cervical rib is probably due to the fact that, as age advances, the posture becomes more

stooped and the muscle tone of the shoulder girdle relaxes allowing the shoulder to fall forward and downward thus giving a greater likelihood of compression.

At the Royal Victoria Hospital since the year 1927, 40 cases of cervical rib have been detected, of which 29 were in females and 11 in males; 25 were bilateral and 15 unilateral; and only half of this number had symptoms. Neurological manifestations preponderated, with 17 showing pain and motor or sensory changes. Three showed pure vascular signs only, while 5 others had a mixed vascular and neurological symptomatology. Fifteen of the 20 patients with symptoms required operation.

The diagnosis of cervical rib is confirmed by x-ray, and surgical treatment consists of section of the scalenus anticus muscle. Resection of a part or the whole rib is in most cases unnecessary. Surgical therapy is, however, reserved for the more severe cases, while physiotherapy designed to improve posture and muscle tone in the shoulder area is frequently sufficient in the milder manifestations.

Scalenus Anticus Syndrome

A study of the anatomical drawing will show the anatomical arrangement of this



Fig 284—X-ray of congenital abnormality of upper dorsal spine with elevation of the right first rib which acts similarly to a cervical rib



A.



B.

Fig 285—A. Normal venogram of the upper extremity.

B Axillary vein compression with stasis due to costoclavicular compression

muscle as it attaches to the first rib anterior to the brachial plexus and subclavian artery. Spasm, or hypertrophy of this muscle narrows the tunnel through which the neurovascular bundle emerges into the arm. This occurs in two ways: the scalenus anticus can cause pressure between itself and the scalenus medius, especially if the latter is abnormally large and more medially placed and, second, subsequent to the muscle spasm the first thoracic rib is elevated causing further compression of the outlet to the arm.

Scalenus anticus syndrome is suspected by the previously mentioned signs and symptoms, local tenderness to pressure over the insertion of the muscle, positive scalene tests and the absence of the other conditions mentioned in the differential diagnosis. An important confirmatory test in both cervical rib and scalenus syndrome is the finding of a systolic bruit over the subclavian artery in the neck which is accentuated during the performance of the Adson test.

The treatment is on the same lines as for cervical rib with the exception that the surgical therapy required is only section of the scalenus anticus muscle (scalenotomy) as close to its insertion into the first rib as possible.

Abnormalities by the First Thoracic Rib

These are uncommon and only occasionally result in symptoms. The abnormality which is concerned here is a broadening and thickening of the lateral portion of the rib over which the neurovascular structures pass. One feature seen in this variety and that of costoclavicular compression is varying degrees of obstruction to the venous return of the arm. Anatomically, the subclavian vein cannot be compressed by a cervical rib or the scalene muscles, but in abnormalities of the first rib or in costoclavicular compression the vein is affected with the neurovascular bundle.

Surgical treatment may require removal of this section of rib or more frequently scalenotomy is all that is necessary.

Costoclavicular Compression

Pressure of the clavicle when pulled downward and backward can cause neurovascular compression against the first rib. This factor is rare and is to be suspected only when typical compression symptoms are present and all other causes have been excluded. Treatment is similar to that of scalenus anticus syndrome. Recently, total resection of the clavicle has been advocated so as to remove the upper compressing bone.

REFERENCES

- Allen, E. V., Barker, N. W., and Hines, E. A., Jr.: *Peripheral Vascular Diseases*, Philadelphia, 1946, W. B. Saunders Company, chaps. 7 and 8.
- Boyd, A. M.: The Diagnosis and Pathogenesis of Obstructive Vascular Disease of the Lower Extremities, *Angiology* 1: 373-390, Oct., 1950.
- Buerger, L.: Thromboangitis Obliterans, *Am. J. M. Sc.* 136: 567-580, Oct., 1908.
- Cohen, S. M.: Traumatic Arterial Spasm, *Guy's Hosp. Rep.* 90: 201-216, 1940-1941.
- Fowler, N. O., Jr.: Thromboembolism: A Survey of the Recent Literature, *Angiology* 1: 257-287, June, 1950.
- Hines, E. A., Jr., and Barker, N. W.: Arteriosclerotic Obliterans: Clinical and Pathologic Study, *Am. J. M. Sc.* 200: 717-730, Dec., 1940.
- Kinmonth, J. B.: Thrombo-angitis Obliterans, Results of Sympathectomy and Prognosis, *Lancet* 2: 717, 1948.
- Luke, J. C.: Arterial Injuries, *McGill M. J.* 15: 251-260, Oct., 1946.
- Luke, J. C.: New Factors in the Etiology of Obstructive Arterial Disease, *Canad. M. A. J.* 56: 377-379, 1947.
- Luke, J. C.: The Diagnosis of Chronic Enlargement of the Leg, *Surg., Gynec. & Obst.* 73: 472-480, Oct., 1941.
- Luke, J. C.: The Costoclavicular Syndrome, *Canad. M. A. J.* 66: 127-131, Feb., 1952.
- Luke, J. C.: Evaluation of the Deep Veins Following Previous Thrombophlebitis, *Arch. Surg.* 61: 787-792, Nov., 1950.
- Mason, M. L., and Weil, A.: Tumor of Subcutaneous Glomus, *Surg., Gynec. & Obst.* 58: 807-816, May, 1934.
- Medical Research Council: Arterial Injuries: Early Diagnosis and Treatment, War Memorandum No. 13, 1944, His Majesty's Stationery Office.
- Pack, G. T., and Miller, T. R.: Hemangiomas, *Angiology* 1: 405-426, Oct., 1950.
- Saland, G.: Acute Occlusions of the Peripheral Arteries: Clinical Analysis and Treatment, *Ann. Int. Med.* 14: 2027-2036, May, 1941.
- Ward, C. E., and Horton, B. T.: Congenital Arteriovenous Fistulas in Children, *J. Pediat.* 16: 746-766, June, 1940.
- Wylie, J., Kerr, E., and Davies, O.: Experimental and Clinical Experiences With the "Clavicle" in the Treatment of a Gravid Abdomen.

CHAPTER XXXIII

THE LYMPHATIC SYSTEM

JOSEPHUS C. LUKE, M.D.

EMBRYOLOGY

The lymphatic system has been described as being the "third system" in the body's fluid transportation mechanism, and is concerned with the return of tissue fluids through various filtration stations back to the general blood stream. The origin of the lymphatics is still not specifically known, but it is generally agreed that the lymphatics primarily develop from the venous system. The histology is very similar, the vessels are valved and, anatomically, the two systems are in close approximation.

According to Sabin, the lymphatic system commences from a series of sacs situated close to the confluence of the principal large veins. From these sacs the lymphatic vessels sprout distally and so form the peripheral lymphatic system. The primary lymph sacs are initially formed from a confluence of venous capillaries and are situated on either side of the base of the neck as the jugular sacs, in the retroperitoneal area as the celiac sac and in each inguinal region as the inguinal sacs. All these become connected, and from them develop not only the lymphatic channels but the principal masses of lymph nodes. The thoracic duct originates from intercommunication between the celiac and left jugular sacs and receives the communications from both iliac (inguinal) areas. The right lymphatic duct develops from the right jugular sac.

An alternate theory is that the lymphatic vessels have a distal origin from the venous system and progress centrally until they fuse into the above-mentioned sacs and intercommunicators. A practical application of lymphatic embryology is the fact that the condition known as cystic hygroma, found al-

most invariably in the neck, is probably the result of a remnant and maldevelopment of one of the jugular sacs.

ANATOMY AND PHYSIOLOGY

Most tissues of the body contain lymphatic capillaries, the exception being those organs or structures which do not contain a normal vascular system. For example, endothelial linings such as the peritoneum or pleura, cartilage, the cornea and the membranous labyrinth do not contain lymphatic vessels. The central nervous system is devoid of lymphatic vessels because the lymph formed drains directly into the cerebrospinal fluid, and in fact composes this fluid. Lymphatic capillaries are numerous in proportion to the blood supply to the part, being, therefore, more frequent in such structures as the skin than in the subcutaneous fat. These capillaries are in general larger than the corresponding vascular ones, are lined by endothelium, have a somewhat beaded appearance, and possess bicuspid valves in the larger radicles.

In the skin the lymphatic capillaries are so numerous that no trauma to the skin or injection into the skin can take place without opening numerous lymphatic channels and so it should be assumed that any injection, be it intradermal, subcutaneous or intramuscular, is absorbed via the lymphatic system. In the leg a somewhat unique construction of lymphatic vessels occurs with two separate systems (the superficial and deep) only connecting at two locations, the popliteal region and fossa ovalis. No intercommunication occurs through the deep fascia except at these points. This fact in

important in an understanding of the relatively frequent condition of lymphedema of the lower extremity.

Situated along the return lymph drainage are strategically placed collections of lymph nodes whose function it is to filter the incoming lymph and so remove foreign material including bacteria. The nodes contain large numbers of phagocytes that play an important part in the destruction of entering bacteria and also are undoubtedly concerned in antibody production. Enlargement of the lymph node occurs during the filtering process (catarrhal lymphadenitis).

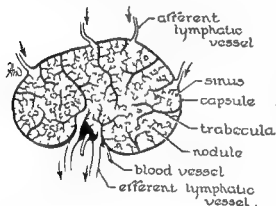


Fig 286—Filtration mechanism of a lymph node

Occasionally the bacterial invasion is sufficiently large or virile that the lymphadenitis goes on to necrosis and abscess formation. Or again if the infection reaching the glands is not too virulent and is continuous though mild in type, a state of chronic hyperplasia and fibrous tissue reaction occurs in the gland and results in permanent palpable nodes (chronic lymphadenitis). These filters can be sufficiently overwhelmed by a virulent infection so that not only does supuration of the node occur but also the infection continues on past one or more nodes to reach the blood stream and so produce a bacteremia or septicemia. It is believed that infection reaches the blood stream almost entirely by the lymphatics, this including the viruses whose passage through the lymph nodes is relatively unobstructed.

It can be seen that a detailed knowledge of the various channels of lymph drainage from an organ or region of the body is of prime surgical importance not only from the point of view of the spread of infection but also for a knowledge of the spread of tumor growth. In the majority of malignant tumors, extension is primarily by the lymphatics and consequently the route of extension can be relatively accurately predicted. Removal of the involved lymphatics and the related nodes is therefore of paramount importance in seeking the cure of a carcinoma.

Lymph Nodes and Vessels

As above indicated, the lymph channels of the lower extremity consist of the superficial and deep sections which communicate only at the popliteal region and through the fossa ovalis in the upper thigh. The superficial channels of the lateral aspect of the foot and leg follow the course of the small saphenous vein emptying into the deep system at the popliteal space. Before entry into the deep channels, the lymph is filtered by two or three popliteal lymph nodes. The medial aspect of the foot and leg and most of the thigh drains into channels which follow the course of the great saphenous vein and join the deep system through the fossa ovalis. Here they pass through the femoral (subinguinal) group of lymph nodes. The corresponding side of the scrotum or labia also drains through the femoral nodes. This group is commonly affected by chronic and acute inflammations in the foot and leg.

Drainage from the buttocks, external genitalia, anal region, vagina and cervix passes to the inguinal glands which are situated parallel and superficial to the inguinal ligament. These glands are frequently involved in venereal infections, skin infections in the drainage area and cancer of the genitalia including the cervix. Both the femoral and inguinal glands drain to the iliac nodes and thence to the main lymphatics of the pelvis, retroperitoneal area and receptaculum chyli.

In the abdomen, the nodes are very numerous both in the mesenteric area and retroperitoneal spaces. The lymphatic vessels and collecting nodes follow the blood supply closely and are located in the mesenteric attachments of the organs. In the stomach the nodes are clustered on either curvature of the stomach, about the lower esophagus and in the region of the first part of the duodenum. Those draining the small intestine are located in the mesenteric leaves and frequently the lymphatic vessels leading to these nodes show as small white threads in the bowel wall due to the milky appearance of the lymph because of the high fat content. This milky lymph (chyle) is also seen in the receptaculum chyli and the thoracic duct, because of the large amount of the lymph collected from the intestinal tract. The degree of milkiness will, of course, vary with the food intake.

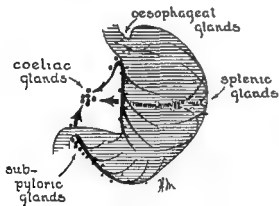


Fig 287—Lymphatic drainage of the stomach.

The hilar region of the lungs and the mediastinum are especially well supplied with lymph nodes which are a mottled greyish black color due to the phagocytosed carbon particles which have reached them from the lungs. Also these glands are frequently the final resting place of lesser degrees of tubercle infection which has entered through the lungs and which has been overpowered at this first line of defense. As in the abdomen, these arrested tuberculous nodes fre-

quently reveal themselves as serrated calcified spots. Efferent drainage from the mediastinal nodes is mainly to the thoracic duct but some from the right lung empties into the right lymphatic duct.

The lymphatic drainage of the breast is of great surgical importance because of the frequency of breast cancer (see Plate VI).

The head and neck have a superficial and deep set of vessels and nodes. The scalp drainage, as in the remainder of the head and neck, is similar on both sides, and the vessels drain sectionally, to specific sets of

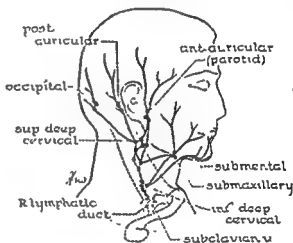


Fig 288—Lymphatic drainage of head and neck.

lymph nodes. The occipital, posterior auricular, and parotid glands receive the scalp lymph, and their efferents connect with the superior deep cervical glands. The lymphatic return from the parotid gland, nasopharynx, mouth, lips and tongue travels through the submaxillary lymph glands, and that of the tip of the tongue and central part of the lower lip and mouth through the submental glands. Further drainage thence is to the superior deep cervical or directly to the inferior deep cervicals and then to the blood stream via the right lymphatic duct on the right or directly into the internal jugular-subclavian vein junction on the left in association with the thoracic duct.

The upper extremity has few lymph nodes in comparison to its bulk. One gland is placed just proximal to the medial epicondyle

and receives the drainage from the ulnar aspect of the forearm and hand including roughly the fourth and fifth fingers. The remainder of the superficial and deep lymphatic drainage progresses directly to the axillary group of glands which lie in the subcutaneous tissues of the axillary space and are readily palpable when enlarged

PHYSIOLOGY

Lymph is a collection of tissue fluid and as such closely resembles blood plasma. The red cell count is understandably low, varying from 500 to 10,000 per cubic millimeter. The white cell (mainly lymphocyte) count is usually double this amount. This is so because of the discharge of lymphocytes into the lymph during its passage through the lymph nodes. The protein content is lower than that of blood, but varies markedly depending on the lymphatic area studied and especially (where thoracic duct lymph is being studied) whether before or after a meal. As a rule, the protein content is about half that of the plasma. The fibrinogen and prothrombin content is low and, consequently, lymph clots, but more slowly than blood. Most of the other constituents such as non-protein nitrogen, chlorides, sugar, urea, uric acid, and calcium have much the same content as the blood.

The lymph pressure and rate of flow are subject to many variables. Studies on thoracic duct fistulas indicate that 1 to 2 cc of lymph are lost per minute. Peripheral lymph has a greater pressure than that in the main channels, the pressure decreasing following passage through a lymph node. The pressure in a normal peripheral vessel rarely exceeds 50 mm of water.

Certain abnormal conditions increase the flow and pressure of lymph. Heat increases production and flow of lymph and, along with the resultant hyperemia, is the reason for the beneficial effect in the treatment of infections. Heat is probably also an accessory factor (in association with prolonged

standing) in producing ankle edema in many persons during the heat of summer. Muscle activity increases lymph flow. Also, any factor producing edema obviously increases production and return flow. Examples of this latter group are increased venous pressures and anything which damages the capillary endothelium allowing increased capillary permeability. This applies particularly to inflammatory edema.

A decrease in the flow and pressure of lymph results from cold and particularly immobility. Congenital malformation of the peripheral lymphatic collecting system such as those probably present in Milroy's disease and lymphedema praecox produce decreased flow and varying degrees of lymph stasis with consequent dilatation of the peripheral lymphatic channels. Surgical removal of main lymphatic trunks, their blockage by cancer cells or infection also give rise to clinical evidence of lymphatic obstruction with edema.

ACUTE LYMPHANGITIS

In acute inflammations the lymphatic vessels are rapidly affected because of the marked number of cutaneous and deep lymphatics, and extension, local or distant, takes place mainly via the lymphatic system. This spread can be of the local diffuse type where the massive cutaneous lymphatic network becomes involved with coagulated infected lymph and cellular debris. The picture is that of a red, warm, tender, edematous area surrounding the local portal of entry and the picture is known clinically as cellulitis. Some degree of cellulitis is always present following a surface infection, but the degree varies. Erysipelas is a type of cellulitis but rapidly extending and massive and the result of streptococci. Local edema is the result of the increased tissue fluid produced by the dilated capillary and the local lymphatic vessel incompetence.

A second type of acute lymphangitis is the so-called tubular variety and the one which is most commonly associated with this term. Reddened, tender, indurated streaks can be

seen ascending to the regional nodes from the point of infection. These are the main cutaneous and subcutaneous lymph vessels which are inflamed and thickened with associated perilymphangitis. Here again the lymph coagulates and becomes mixed with inflammatory debris. This type of lymphangitis has been known by the lay public as blood poisoning and is still occasionally referred to by this term. This name is based on fact as lymphatic infection either of the local or tubular type extends rapidly and soon gives generalized signs and symptoms of infection. Fever up to 104° F., chills, general malaise and nausea with vomiting are not uncommon. Presumably the organisms have passed through the lymph nodes and have reached the general circulation. Formerly septicemia and death were all too common sequelae.

At the present time the antibiotic drugs have completely changed the picture of acute lymphangitis. A fatal outcome is now unknown, the inflammatory picture rapidly subsides and complete restitution to normal takes place in a few days. During the initial acute phase, the patient should be at rest in bed, the local area also at rest and enclosed with hot, moist dressings. Evidence of the collection of pus either at the site of entrance or along the lymphatic pathways should be watched for and drained. Late neglected cases show, besides systemic effects, local penetration by the infection of neighboring structures. Osteomyelitis, tenosynovitis or fascial space abscess are possible results.

CHRONIC LYMPHANGITIS

Chronic lymphatic infection from a subacute focus such as athlete's foot infection or chronic septic tonsils is a frequent occurrence. Also recurrent acute attacks of cellulitis and lymphangitis leave progressively increasing damage to the collecting lymphatic channels. The inflammation of the lymphatics and the presence of infected coagulated lymph leave, following its resolu-

tion, a gradually increasing impairment of the function due to increasing scar tissue formation. The whole picture is that of a progressively increasing degree of stenosing lymphangitis. Such a result appears to take several months of chronic involvement or many recurrent attacks of acute lymphangitis before severe degrees of lymphatic obstruction occur, and it is unlikely that in the future the predisposing conditions of stenosing lymphangitis will be marked because of the widespread use of the antibiotics. However, at present we are dealing with the cases of chronic lymphatic obstruction caused by the lymphatic insult of many years ago.

TYPES

1. Recurrent attacks of erysipelas or erysipeloid infections give gradually increasing degrees of lymphatic vessel obstruction due to scar. It is this type which results in the severe degree of elephantiasis occasionally seen in those who have never been in the tropics and where filariasis is not a factor. This is rarely of importance except in the lower extremity.

2. Filariasis of the Bancrofti type tends to settle in the lymphatic system and is especially destructive to the lymph nodes of the lower extremity. Contact with this worm can occur via any break in the skin, and the spread is primarily by the lymphatic system. Subsequent involvement of the blood stream may take place. The inguinal and femoral lymph nodes become involved by the irritative effects of the filaria, undoubtedly also aggravated by the presence of the secondary bacterial invaders. A stenosing lymphangitis and lymphadenitis is set up with effects of severe lymphedema similar to that seen following the streptococcal variety. Scrotal edema is also commonly associated.

3. Acute thrombophlebitis of the large veins of the thigh and pelvis is invariably associated with perivenous inflammation and hence later scar distortion of the larger contiguous main lymphatic trunks from the legs. Chronic lymphedema results, aggra-

vated by the presence also of chronic venous stasis from the defective deep venous return. The combination of these two factors results in the disabling complications of the post-phlebitic leg. (See pages 653-657 for further details of chronic lymphedema)

LYMPHADENITIS

Acute Lymphadenitis

Infected lymph from a superficial area passes through the appropriate lymph node in its passage centrally. In the gland an attempt is made to combat the invading organism. There are gross enlargement, tenderness, and hyperemia of the gland and the microscopic picture is that of marked proliferation of lymphocytes, and also of the endothelial cells. Hyperemia and edema take place along with considerable periadenitis. If the infection is conquered, complete restitution to normal occurs or, if not, the defense forces in the gland are overcome, leukocytes appear, pus is formed which eventually breaks into the surrounding tissues to form an abscess. The commonest location for these nonspecific types of lymphadenitis is in the submaxillary gland group following tonsillar and mouth infections, femoral node abscesses from infection in the feet and axillary node involvement from the hand.

Treatment consists mainly in controlling the local infection which gave rise to the lymphangitis and lymphadenitis. The antibiotics, as previously mentioned, have markedly reduced the incidence of this condition and have led to an early resolution. Rest and heat to the inflamed gland masses are indicated.

Chronic Lymphadenitis (Nonspecific)

Repeated lesser degrees of inflammation draining to lymph nodes set up a subacute lymphadenitis which does not go on to suppuration but results in chronic enlargement of the glands. Microscopically, increased lymphocytic content is present along with

considerable fibrosis. Elastic enlarged nodes result which rarely completely resolve unless the primary focus is removed. The most common examples of this type are seen in the femoral nodes following chronic epidermophyton infection between the toes, the inguinal nodes from chronic infection in the anal and genital area, and in the neck from foci in the nose and throat.

From the therapeutic standpoint these glands are of little importance except to point up a chronic focus of infection. Relief of the focus will cure the enlarged glands. One important point is the similarity between this condition and the clinical appearance of the early case of tuberculous lymphadenitis.

Specific Forms of Lymphadenitis

(A) *Tuberculous Lymphadenitis*.—This form of lymph node involvement is much less frequent than formerly in North America because of the strict control of the public sale of milk. Pasteurization has reduced this disease to a minimum, especially among city dwellers, and the cases now seen usually come from the country in areas where pasteurization is not used. In England, Scotland, and Wales less stringent milk control still produces a large number of gland, bone, and joint tuberculosis.

Tuberculous adenitis in the child is almost invariably the result of the bovine type of bacillus acquired from infected milk, but in the occasional adult case the organism can be either bovine or human in type. The lymph glands of the neck are most frequently involved, the germs entering the lymphatic system from a tonsillar focus or through one of the pharyngeal lymph plaques, infected tooth or other break in the lining of the oral cavity. The organism can come via infected milk or sputum coughed up from a pulmonary lesion.

The mesenteric lymph nodes are next most commonly involved, becoming infected from organisms absorbed from the gastrointestinal tract originating either in infected food and

drinks or the swallowing of tuberculous sputum. The ileocecal region is the most frequent portion of the gastrointestinal tract to be involved. An x-ray of the abdomen taken for other reasons frequently shows an irregular, roundish patch of calcification about 1.25 cm. in diameter. These are tuberculous glands where the process has finally been conquered but not before gland destruction and later calcification of the caseated gland have occurred. This finding can be associated with no past or present signs or symptoms of the disease, and such a finding is common also in routine chest radiography.

Nodes draining one of the extremities are rarely involved by tuberculosis unless the organism gains entrance to the distal tissues through a small abrasion. Such an accident is most common in persons such as nurses whose work brings them in contact with open cases of tuberculosis. A localized, firm, tender, painful nodule develops in the skin at the site of accidental entry of the germ. A small, shallow, painful ulcer usually develops at this site, and later enlarged, tender regional nodes appear. The local finger lesion was formerly known as a butcher's wart because of the frequency of this condition in those who handled infected meat.

Pathology.—When the bacillus of tuberculosis is conveyed to a lymph node by the lymphatics, the reaction in the node depends on the degree of invasion and the resistance of the host to the invader. All degrees of node response may occur from single node hyperplasia to that of caseation and abscess formation. In the lesser degrees, typical tubercle formation may be hard to find and the determination of the correct diagnosis may be difficult even on pathological section and the diagnosis is easily mistaken for chronic non-specific lymphadenitis.

Clinically, the glands are initially discrete and elastic but later become matted into masses due to the development of peradenitis. An actively infected or caseating node may rupture into the blood stream and re-

sult in the miliary generalized form of the disease. In the more advanced cases tubercles are numerous, areas of caseation are obvious, and the typical "cold abscess" of tuberculosis may result. Incision and drainage of such an abscess under the mistaken impression that it is due to pyogenic organisms invariably produce a fistula which remains as a discharging sinus indefinitely because of the underlying infected gland and the inevitable secondary infection incident to the draining wound.

The moral to be emphasized in this connection is to suspect all abscesses which have developed in a subacute manner, especially in the neck. In the tuberculous case, the constitutional reaction is minimal, the origin of the infection is obscure, and the abscess shows minimal signs of inflammation, tenderness, and surrounding induration. When doubt exists, the pus should be aspirated and cultured, and if tubercle bacilli are found, then incision of the infected abscess is indicated with removal by curettage of the underlying infected gland or glands.

Formerly, extensive surgery including block dissection of all the neck nodes was the accepted procedure in tuberculous adenitis, but at present such an extensive degree of the disease requiring such treatment is rarely seen. Improvement in the medical management has also decreased the need for surgery. Control of the milk supply, removal of infected tonsils, improvement in general health subsequent to adequate rest, nourishment, vitamins and heliotherapy will result in cure in most instances. As indicated above, the role of surgery in tuberculous adenitis is the evacuation of abscesses and the local removal of caseating abscessed nodes. Streptomycin is the most recent successful aid to treatment.

Syphilis.—Involvement of lymph nodes is seen in both the primary and secondary stages of syphilis. In the former, the nodes adjacent to the chancre become involved in 5 to 14 days following inoculation. The inguinal glands are, therefore, most commonly

involved and the glands present as firm, discrete, tender nodules. The adenitis is due to a combination of the *Spirochaeta pallida* and other secondary invaders from the chancre. The diagnosis is not difficult when the chancre is seen and is confirmed by dark-field examination of the secretion. The secondary stage of syphilis is frequently associated with a generalized lymphadenopathy, the glands again being discrete, rubbery, nontender and do not go on to suppuration. Tertiary gumma in lymph nodes is extremely rare and will be less common in the future because of the decrease in incidence, improved detection, and improved treatment of this disease.

Lymphogranuloma Venereum (Lymphogranuloma inguinale).—This condition has been called the fourth venereal disease and is caused by a filtrable virus. The genitalia are the most common site of entry, but of course digital and oral origins can occur. The initial surface lesion rarely is as large as a chancre and may be so insignificant as to go unnoticed. Several weeks after contamination, the regional nodes become swollen, indurated, and painful. Necrosis of the gland results and suppuration occurs. This pus tends to burrow to the surface or surrounding structures unless aspiration is carried out. In the inguinal region, ulceration and sinus formation with later extensive scarring are the usual sequelae. In some cases extension to the perirectal tissues with perineal or rectal fistula takes place. The incidental scarring subsequent to this type of spread may result in extensive stricture formation of the lower rectum. Such a sequel appears to be more common in females and these strictures were formerly supposed to be syphilitic in origin because the pathological picture is that of a nonspecific granuloma.

The diagnosis should be suspected from the clinical picture of the inguinal bubos or when rectal stricture is present. Carcinoma and posthemorrhoidectomy strictures should be excluded. The Frei test gives confirma-

tion to the diagnosis and consists of an intradermal injection of diluted pus from one of the lesions which has been autoclaved at 56° C. A positive test is indicated by a central papule at the site of injection with more than a 1 cm. ring of surrounding erythema. Treatment consists of aspiration of abscesses and the use of massive doses of aureomycin and penicillin.

Mesenteric Lymphadenitis

The mesentery of the gastrointestinal tract is plentifully supplied with lymph vessels and glands. Involvement of those glands due to infection absorbed from the intestine is not uncommon. Enteritis of different types invariably produces lymphadenitis, but fortunately this is rarely of clinical significance because of its transient nature. Two forms of nonspecific mesenteric adenitis merit description.

Acute Mesenteric Lymphadenitis.—In this variety, the glands in the ileocecal angle are most frequently involved, the primary focus being either in the lymphoid tissue of the appendix or terminal ileum. The condition is most commonly seen in children and is manifested by pain, splinting and tenderness in the right lower quadrant associated commonly with nausea and vomiting. The associated fever and leukocytosis are usually higher than with appendicitis. Apart from this, the clinical picture is indistinguishable from that of acute appendicitis and consequently the diagnosis is usually made at operation done for a supposedly acute appendix. Red, swollen, and markedly enlarged glands up to the size of a walnut are found with little evidence of any inflammation of the terminal ileum or appendix. Rarely does suppuration with abscess formation occur and the usual outcome is rapid subsidence of the condition following appendectomy and the use of the antibiotic drugs.

Chronic Mesenteric Lymphadenitis.—In common with all lymphatic tissue, the lymph glands in childhood and adolescence are

more hyperplastic than in later adult life. The mesenteric glands share in this involvement and, as a result, produce symptoms due to their rigidity where mesenteric movement is involved. The patient is usually a child between the ages of three and thirteen who complains of intermittent spasms of abdominal pain. The spasms may be momentary or persist up to fifteen to thirty minutes. The pain is not typical colic, but is described as an intermittent knifelike pain centered about the umbilical region. Nausea, vomiting, and anorexia are occasionally associated. Vague tenderness is present in the right lower quadrant or mid-abdomen, but splinting is minimal or absent. A mild degree of fever occurs in most cases but the leukocyte count is little if any elevated. The local point of tenderness found either in the right lower quadrant or subumbilical region frequently shifts to the left when the patient is turned on the left side (Brennan's sign). The significance of this is the positional shift of the mesentery with its offending enlarged glands. Such a shift does not take place when the cecum and appendix are involved.

The diagnosis is difficult to distinguish from chronic appendicitis but the above-mentioned points should tend to indicate the possibility of chronic mesenteric adenitis. Active therapy is necessary only if symptoms persist and interfere with the activities and nutrition of the child. Normally the complaints will disappear with time, especially after puberty. However, when of a disabling nature or when chronic or subacute appendicitis cannot be excluded, then appendectomy is indicated.

On opening the abdomen a somewhat increased quantity of straw-colored peritoneal fluid is encountered. The lymph glands in the small bowel mesentery are numerous and enlarged up to the size of a bean. Biopsy of one of these glands invariably shows a catarrhal lymphadenitis. Removal of the relatively normal-looking appendix is all that is indicated in these cases and appears to bring about a complete regression of signs

and symptoms in most cases. Formerly it was considered that these enlarged lymph nodes were a chronic hyperplastic form of tuberculous adenitis, but pathological examination of many glands removed for biopsy failed to confirm this idea, and also bacteriological study of the removed gland always fails to yield any growth.

HODGKIN'S DISEASE

In 1832 Hodgkin described a series of glandular swellings associated with splenomegaly and made the first description of the disease which bears his name. It is likely that he described other lesions in his original paper, but four of the cases appear to fit closely with what we now know as Hodgkin's disease. The etiology of this condition has not been clearly settled because it shows not only many of the features of an infective granulomatous lesion but also those of a malignant tumor, being particularly invasive in its terminal stages.

Jackson and Parker have made a differentiation of varying stages of this disease. They have called the initial stage *Hodgkin's paragranuloma* indicating that the pathological picture is close to but not typical of Hodgkin's. This is an important differentiation because of the pathological difficulties in labelling a case one of true Hodgkin's disease. The author has several cases where a pathological probable diagnosis of Hodgkin's disease has been made and a poor prognosis given only to have the patient continue in good health or with further episodes of symptomless glandular enlargements for more than fifteen years. It is obvious that such longevity indicates features not in keeping with true Hodgkin's disease where the prognosis is fatal in one to four years. This latter fatal group is called, by Jackson and Parker, *Hodgkin's granuloma*. The malignant, invasive variety of Hodgkin's disease which appears to progress from the granulomatous stage has been called, by Ewing, *Hodgkin's sarcoma* and appears mainly in the older age group. Therefore, it would appear wise to divide this disease into the

three above stages, each of which has to be settled by a combination of clinical and pathological criteria

The sex incidence appears higher in males (3:1) and the disease may occur at any age. The paraganuloma is seen mainly in post-pubertal adults, the granuloma in the early adult age, and the sarcomatous type in the older age groups. The patient usually presents himself with a gradually progressive enlargement of various lymph nodes particularly those of the neck. In the early stages (paraganuloma and early granuloma stage) the constitutional symptoms are absent but later weight loss, anorexia, anemia and pressure symptoms from the gland masses are present. An unusual variety of unexplained fever (Pel-Ebstein) is occasionally associated with this disease. The temperature rises over a few days to a level of 101° to 103° and is maintained at this level with minor variations for 7 to 14 days after which it returns to normal. Recurrent bouts of this pyrexia usually occur.

The spleen is moderately enlarged in about 70% of the cases, and blood examination shows, in the later stages, a definite anemia, occasionally eosinophilia, and an increase in large mononuclear leukocytes. These blood changes are seldom sufficiently characteristic to be diagnostic. Primary involvement of the gastrointestinal tract can occur.

Pathological Appearance.—The lymph glands are enlarged but remain discrete and rubbery until the late stages. They have an elastic consistency unlike the rocky hardness of secondary cancer deposits in glands. On section the gland shows a grey, translucent appearance, markedly different from the encephaloid appearance of secondary cancer. In microscopic section, the characteristic points are the replacement of the normal lymphoid tissue by a multitude of different cell types. The most prominent of these is a large, pale epithelioid cell but most characteristic are the giant cells. The latter are mono- or multinucleated and are called after their discoverers, Reed and

Sternberg. This characteristic cell is found both in the paraganuloma and the granuloma. Other cells present include eosinophils, lymphocytes, mono- and polymorphonuclear leukocytes. The above picture is also seen scattered in other organs especially the spleen, liver, and bone marrow. Hodgkin's sarcoma usually shows direct extension of the disease into lung, bone, liver, and portions of the gastrointestinal tract.

Treatment is a combination of surgery and radiotherapy. Surgery is indicated mainly for the purpose of obtaining biopsy material, but it is wise to remove the entire enlarged gland at time of operation rather than a mere portion of the gland. X-ray therapy produces remarkable regression of the enlarged glands and is used in different locations as new gland masses arise. In the paraganuloma, the regression following x-ray therapy may last for years but once the disease is well established, the benefit from this therapy is only temporary as other manifestations soon arise. However, there is no doubt that x-ray therapy is the best therapeutic agent available, but unfortunately, recurrent enlargement of the original nodes following x-ray responds less favorably on secondary irradiation. Teroplerin and nitrogen mustard may be useful in the terminal stage to give some alleviation of symptoms but are purely palliative.

LYMPHOSARCOMA (LYMPHOBLASTOMA)

This very fatal disease can be considered a primary neoplasm of lymphatic tissue. It has the characteristic of appearing in scattered areas of lymphatic tissue almost simultaneously. There is considerable clinical resemblance between this form of lymphatic abnormality and that of Hodgkin's disease as the initial complaint is that of a lump in one of the lymph gland areas. As in Hodgkin's disease the neck is the commonest site of origin although the groin and axilla are also frequently primarily involved. The individual lymph glands rapidly become fused



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Plate XLIV.—Hodgkin's Disease.

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from local extension of the tumor and even may ulcerate through the skin.

There are no specific symptoms of the disease, the patient seeking attention because of a lump or symptoms the result of pressure and involvement of surrounding structures. The spleen may be found to be somewhat enlarged. Similar to Hodgkin's disease, the blood changes are not characteristic but usually show a leukocytosis which can be caused by either an increased lymphocyte or polymorphonuclear count. In the former type, confusion may result from the resemblance to lymphatic leukemia. Anemia and some fever are frequently associated with the late cases and generalized metastases may be spread throughout solid organs. Lymphatic leukemia may develop in a case of lymphosarcoma and vice versa, especially in childhood where the greatest incidence of the disease takes place.

Not only lymph glands but other lymphoid structures are primarily the seat of the disease. These include the tonsils, the adenoids and the gastrointestinal lymphoid tissue. In the gastrointestinal tract the stomach is the organ most commonly affected and the preoperative clinical x-ray picture is indistinguishable from carcinoma. The ileum with Peyer's patches may also show the first signs of the disease, while the rectum and the colon are next in frequency. At the Royal Victoria Hospital 23 cases of primary lymphosarcoma of the gastrointestinal tract were found from a total of 100 cases of this disease.

Cases of Lymphosarcoma.—

| | |
|---------------------------|-----|
| Generalized spread | 56 |
| Primarily retroperitoneal | 21 |
| Gastrointestinal | 23 |
| Stomach | 6 |
| Duodenum | 3 |
| Jejunum | 2 |
| Ileum | 3 |
| Cecum | 3 |
| Colon—other parts | 3 |
| Rectum | 3 |
| Total number of cases | 100 |

Pathological Features.—The gross appearance of the cut gland is not characteristic. It is whitish grey to pink in color and is softer than the corresponding gland of Hodgkin's disease. Fusion of contiguous nodes occurs early in the disease.

Microscopically, two main varieties of pathological types are distinguished. The more common is that where the gland appears to have been replaced in its architecture by a diffuse collection of small round cells resembling small lymphocytes. Occasionally a larger type of lymphocyte is seen similar to the appearance of a lymphoblast. This variety shows a uniform pathological appearance differing markedly from the pleomorphic cell types of Hodgkin's disease. Giant cells do not occur in lymphosarcoma. In the second variety, the reticulo-endothelial elements appear to be primarily involved and consequently it has received the name of a *reticulum cell sarcoma*. It is argued by some authorities that this is not a variety of lymphosarcoma but a separate tumor which arises from the reticulo-endothelial system of lymph glands. Especially does this concept hold when it is known that this type of tumor can be primary in bone and other tissue where lymphatic tissue is minimal. In the lymph gland, the structure is destroyed and is replaced by large, pale staining cells of endothelial type, arranged loosely and with a more pronounced reticulum than in the lymphoid type.

It must be borne in mind that diagnosis of one of the lymphomas on pathological appearance only may contain a margin of error because of the considerable variations in the picture from case to case. At times, a simple hyperplastic lymph node may be difficult to differentiate from the lymphatic variety of lymphosarcoma, consequently diagnosis should be made in combination with a suggestive clinical picture and a progressive clinical course. It is likely that cases of lymphosarcoma that survive many years have not been authentic instances of the disease.

and is known as *malignant thymoma*. The epithelial cell type is a true *carcinoma* arising from Hassall's corpuscles. Both varieties are highly malignant and rapidly fatal, yielding only briefly to x-ray therapy. Local compression and invasion of the chest structures and lymphatic node spread occur early. General metastases sometimes are found.

PERIPHERAL ABNORMALITIES OF THE LYMPHATIC SYSTEM

Tumors of Lymphatic Origin

Tumors of the lymphatic system are relatively rare if the dilated lymph spaces associated with acquired or congenital lymphedema are excepted. True lymphatic tumors can be divided into the simple, the cavernous and the cystic. The simple *lymphangiomas* are usually congenital and are manifest as small warty tumors on the skin or mucous membranes. They are elastic to the feel and are white to pink in color and consist of a meshwork of dilated lymph spaces and lymph vessels set in a thin reticular network. The *cavernous type* is also congenital in origin and is frequently combined with hemangiomatous tissue. Large areas of the skin and subcutaneous tissue may be involved and result in a tumor-like mass of dilated vesicles containing lymph with a spongy mass of greyish tissue in which are mingled spots of hemangiomatous tissue. These tumors are most commonly seen in the upper extremity and shoulder region, the lips, eyelids and tongue. Surgical excision is the best therapy, but recurrence will take place if not totally removed. X-ray and radium can be used, but are probably most effective where excision has not been total or where recurrence has taken place.

Cystic lymphangioma (cystic hygroma) is also congenital and develops from endothelial cell rests remaining from the formation of the embryonic jugular sacs from which the lymphatics of the head, neck, and arms develop. They are most common at the base

of the neck but may occur in the situations of the other primary embryonal lymph sacs (inguinal and celiac). Cystic hygromas may be well developed at birth and cause dystocia or may develop in early childhood. The typical one is a large, soft, cystic swelling in the supraclavicular area, lobulated and containing large quantities of lymph. Well-marked transillumination occurs. In common with other lymphatic collections, they become infected easily, and treatment should be carried out early to prevent this complication. Radical surgical excision is the best therapy.

LYMPHEDEMA

The development of the lymph vessels is closely akin to that of the veins and they resemble veins in being endothelial lined tubes containing valves. They closely follow the main vascular channels. The lymph in these channels is strained through various sets of lymph nodes and is collected in the main lymph ducts which empty into the venous system. In consequence of their similarity to the venous system, lymphatic vessels are heir to many of the abnormalities which affect veins. In the extremities there are two separate sets of lymph channels, those of the superficial tissues and those of the deep. In the leg, the superficial ones follow the great and small saphenous veins and communicate with the deep vessels only in the popliteal and groin areas.

Lymphedema is most common in the lower extremity due to the upright posture and the frequent anomalies of the vessel walls and valves which occur. In this respect, the parallel with the venous system is marked, one producing lymphedema and the other varicose veins. Any abnormality which results in lymphatic obstruction or stasis can result in lymphedema. Such abnormalities are manifested mainly in the superficial lymphatics because of the lack of support in the subcutaneous tissues. These derangements include congenital weakness of lymphatic walls and valves, block dissec-

Treatment is similar to Hodgkin's disease with surgery being limited to the obtaining of material for biopsy. Radiation therapy produces a rapid melting away of the glands and relief of symptoms, but, unfortunately, the disease usually recurs in other areas. Radiation therapy can be used as a diagnostic test, especially in those cases where the mediastinal glands are involved. The obtaining of biopsy material is not practical in such cases, and if the symptoms rapidly decrease and the chest picture rapidly improves under treatment, it is likely that lymphosarcoma is the cause of the glandular enlargement. As in Hodgkin's disease, nitrogen mustard is of some palliative benefit in the advanced cases. Cortisone and ACTH therapy has so far been disappointing in the treatment of lymphoma.

BRILL'S GIANT FOLLICULAR HYPERPLASIA

(Giant Follicle Lymphoma)

This variety of lymph gland enlargement is of importance because of its potentialities. Pathologically it is characterized by the large number of hypertrophied lymphoid follicles seen in the gland. These can often be seen grossly in the freshly cut gland. The enlarged follicles are packed with actively proliferating lymphocytes sufficiently neoplastic as to invade the gland capsule. As a rule only a few glands are involved, and it is rarely that the disease becomes generalized.

The importance of this variety of lymphoma is the frequency of its progression (about 65%) to one of the more malignant lymphomas. Lymphosarcoma, either lymphocytic or reticulum cell type, is the most frequent, but Hodgkin's disease has also been reported to arise from this focus. Radiation therapy is the best treatment, but constant observation must be made for possible future developments.

THE THYMUS

Although generally considered a portion of the lymphatic system, the thymus is composed of two separate elements. The medulla is derived from the fourth branchial cleft and is composed of epithelial cells derived from the cleft. These are mainly collected in cell aggregations known as Hassall's corpuscles whose function is not fully known. The thymic cortex is composed mainly of lymphocytes and the variations in size of the organ during disease states and in varying ages are mainly due to the increase or decrease of the lymphocyte content.

In disease states, the thymus decreases in size due to loss of lymphocytes, whereas in sudden death the gland size corresponds to the normal. This latter finding was formerly mistaken to indicate a pathological increase in the size of the gland especially in infants and children with sudden unexplained death. The terms *status thymolympathicus* and *thymic death* were coined in an attempt to describe these cases, but it has been shown that these so-called enlarged thymus glands were either normal or little enlarged. At birth the gland weighs about 13 grams and increases to 35 grams at puberty, gradually becoming atrophied in adult life. The thymus rarely is sufficiently enlarged to produce direct pressure symptoms on the trachea, and therefore should be kept in mind in cases of unexplained moderate dyspnea. X-ray including anteroposterior and lateral projections will give the clue to such a condition. In diseases which result in lymphatic hypertrophy, the thymus is also enlarged, including lymphatic leukemia and Graves' disease.

Tumors

As the thymus consists of the two separate elements, cortex and medulla, it is heir to rare tumors involving the cortical lymphatic tissue or the epithelial-celled medulla. The former type resembles lymphosarcoma either of the lymphocytic or reticulum-celled type.

this enlargement originally disappearing with elevation of the limb, but later, due to the increased lymphatic enlargement and the accompanying fibrosis, the enlargement only partially subsides on elevation. The patient originally reports for cosmetic reasons, but later complains of tiredness, heaviness, and aching of the enlarged leg. Subsequent waves of erysipeloid-like inflammation of this lymph-soaked tissue may result.

tioned above. The degree of lymphedema will depend on the extent of this obliteration and resembles the hereditary variety in being confined to the skin and subcutaneous tissue. The swollen arm after radical mastectomy for carcinoma is the result of lymphatic vessel obliteration by carcinoma, chronic infection, x-ray or radical removal of the pathways, while a swollen leg is a late complication of extension of carcinoma

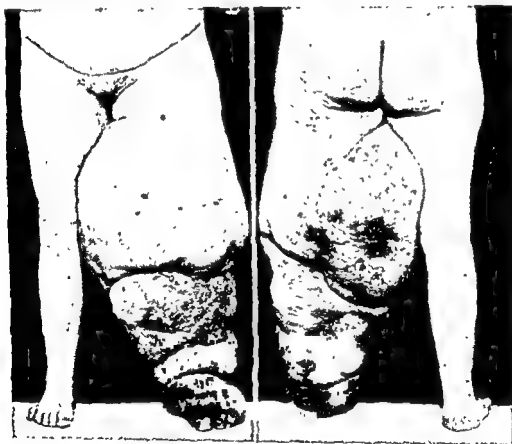


Fig 291.—Severe degree of elephantiasis resulting from chronic stenosing lymphangitis and lymphadenitis.

Milroy's disease is almost identical with lymphedema praecox, but, to conform with the original description of Milroy, it must be both hereditary as well as congenital and therefore present in other members of the same family

Obstructive Lymphedema

Actual occlusion of lymphatic pathways can be produced in the various ways men-

tioned above. The degree of edema seen in the stenosing lesions caused by cancer or infection.

One of the more common causes of lymphedema of the acquired type is a stenosing lymphangitis and lymphadenitis, the result of multiple attacks of lymphangitis usually produced by the streptococcus. The

tion of lymph nodes and channels, x-ray obliteration of these channels or their occlusion by neoplastic cells or fibrosis following multiple waves of lymphangitis and lymphadenitis. This occlusion and subsequent stasis result in dilatation of the distal lymph spaces to all degrees from mild swelling to the huge leg of elephantiasis. The collection of lymph in numerous dilated lymph



Fig 289—Lymphedema praecox, left leg, in woman aged 20 years

channels in an extremity results in gradually progressing fibrosis as the high protein content of the lymph is a powerful stimulant to fibroblastic proliferation. This fibrosis in an extreme degree produces the warty excrescences, the thickened inelastic skin and doughy subcutaneous tissue of the elephantine leg seen in long-standing lymphedema. Infection in this static lymph is also a frequent accompaniment, the streptococcus being the most common organism. Recurrent attacks of cellulitis develop, each of which causes further lymphatic obstruction by its associated lymphangitis and lymphadenitis

Lymphedema Praecox

This is a variety of lymphedema of the leg and rarely of the arm which comes on most frequently about the time of puberty but may be seen either earlier or later than this period. Females are more commonly affected than males. A gradual enlargement of one leg, and possibly later the other, occurs beginning in the foot and ankle and slowly progressing up the leg. The origin is probably analogous to that of varicose veins where a congenital deficiency of the lymphatic system is present involving the lymph valves and possibly the vessel walls. These defective vessels gradually fail to function normally, and dilatation of the lymph vessels to lymph sacs occurs in the most dependent part of the limb. Lymph stasis with enlargement of the limb occurs,



Fig 290—Lymphedema praecox in man aged 26 years

trolled by conservative measures, then surgical therapy is indicated. This consists of the Kondoleon procedure and its modifications which remove as much as possible of the lymph-soaked skin, subcutaneous tissue, and deep fascia of the limb. This can usually be done in two or three stages to complete the circumference of the leg. The original plan in this procedure was to establish a communication between the superficial and deep lymphatics whereby the former could drain into the latter by the development of new channels made possible by removal of the deep fascia. However, the major benefit probably results from the mechanical removal of the lymphedematous tissue. Following this method, a strong elastic stocking must still be worn to prevent swelling, as the original reason for the blockage still persists

REFERENCES

- Best, C. H., and Taylor, N. B.: *The Physiological Basis of Medical Practice: A Text in Applied Physiology*, ed. 5, Baltimore, 1950, The Williams & Wilkins Company.
- Drinker, C. K., and Field, M. E.: *Lymphatics, Lymph and Tissue Fluid*, Baltimore, 1933, The Williams & Wilkins Company.
- Drinker, C. K., and Yoffey, J. M.: *Lymphatics, Lymph and Lymphoid Tissue*, Cambridge, 1941, Harvard University Press.
- Ewing, James: *Neoplastic Diseases; a Treatise on Tumors*, ed. 3, Philadelphia, 1928, W. B. Saunders Company.
- Hodgkin, T.: On Some Morbid Appearances of Absorbent Glands and Spleen, Royal Medical and Chirurgical Society of London, *Medico-Chirurgical Transactions* 17: 68-114, 1832.
- Jackson, Henry, Jr., and Parker, Frederic, Jr.: *Hodgkin's Disease and Allied Disorders*, New York, 1947, Oxford University Press.
- McMaster, P. D.: Lymphatics and Lymph Flair in Edematous Skin of Human Beings With Cardiac and Renal Disease, *J. Exper. Med.* 65: 373-392, 1937.
- Sabin, F. R.: The Method of Growth of the Lymphatic System, *Harvey Lecture Series* 11: 124-145, 1915-1916.

history of such a case is of many attacks of erysipeloid infection in the extremity associated with chills, fever, and general malaise. Each attack is followed by increasing degrees of lymphedema in the limb. This chronically edematous limb appears to harbor the offending organism in a quiescent form between attacks, and the infection is reactivated by more than usual overuse of the leg. This variety of lymphatic stenosis leads to the immense legs of elephantiasis.



Fig 292—Elephantiasis resulting from multiple previous attacks of leg erysipelas and associated lymphatic blockage

Swelling of an extremity due to lymphedema has several characteristics. The enlargement is usually unilateral and the swelling subsides only slightly on recumbency in the well-established case. The extremity is whitish in color and the edema is doughy and pits only on prolonged pressure. Varicose veins are absent and there is no history of previous involvement of the deep venous system. The arterial circulation is normal. The symptoms are those due to the result of the increased size and weight of the limb.

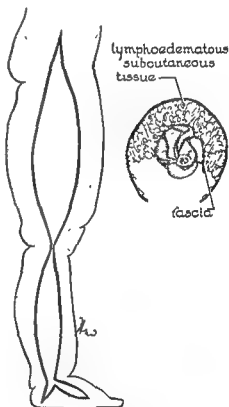


Fig 293—Kondoleon procedure for severe degrees of lymphedema.

The word *elephantiasis* is purely a descriptive term and does not denote the etiology. In tropical climates elephantiasis is more common due to the *filaria Bancrofti* which settle mainly in lymph channels and glands and set up a chronic irritation (frequently in association with the streptococcus) and so produce an extreme degree of lymphatic occlusion.

No reliable method has yet been devised to compensate for blockage of the lymphatic return. Consequently treatment is mainly palliative and consists, in the early stages, of elevation of the limb as much as possible and the wearing of compression bandages, the best being a strong elastic stocking or strong elastic or rubber bandage. If the degree of swelling is great and is not con-

tensive on the surface than in the deeper tissues. Extreme conservatism is indicated to allow a proper line of demarcation to develop. After the dead tissue is removed, the use of suitable skin grafts will save many a foot or hand which originally appeared doomed.

In those cases where amputation is indicated because of deficient blood supply, the result of main artery occlusion, no hard and fast rules can be laid down as to the proper level of amputation. The fact should be kept in mind that amputation is being done for an arterial lesion which is usually progressive and that a successful low amputation may break down months hence because of progression of the disease. This observation applies especially to Buerger's disease. Another point to be remembered is that a lower level stump, though healed, may not be sufficiently supplied with blood to withstand the trauma incident to wearing an artificial limb. Each case is individual and a careful preoperative study is necessary to determine the efficiency of the existing collateral flow and to ascertain whether this flow can be improved by suitable measures.

It is our practice to carry out the following investigations prior to every amputation indicated because of occlusive arterial disease: Palpation of the pedal and leg arteries is of extreme importance, because it can be stated somewhat dogmatically that primary intention healing (in the absence of infection) will take place after toe amputations if the dorsalis pedis and posterior tibial arteries are felt to pulsate, and almost invariably if only one of the two is actively functioning. Similar findings apply to a transmetatarsal amputation. Healing of a below-knee site of election amputation will take place when the popliteal artery is found to be patent and a good result will follow a lower third of thigh amputation when the common femoral artery is patent.

Frequently a lower amputation can be done successfully in the absence of the above criteria where it can be shown by symp-

thetic nerve block that the circulation at the proposed amputation level is capable of further dilatation, this being indicated by increased skin temperature readings. Where such improvement is seen, a lumbar sympathectomy done at the time of amputation will ensure a successful lower level of amputation. The histamine flare test and the use of intravenous fluorescein are also aids in the determination of the level of adequate collateral circulation and therefore successful healing.

In gas gangrene, amputation levels should be above the area of visible diseased tissue. If, at operation, certain muscle groups are found to be involved higher than was anticipated, these groups can be excised and so allow for a longer stump than otherwise would be the case.

In limb sarcoma, the level should be as high as is practicable in order to reduce the possibility of further extension but it is unfortunately too common that distant metastases have already occurred via the blood stream before amputation is done. A mid-thigh amputation is indicated for growths involving the foot and lower leg or hip disarticulation in tumors of the lower femur. The same levels apply to the upper extremity and a fore quarter amputation should be carried out for the neoplasms involving the shoulder region or hind quarter ablation in those involving the hip area.

REFRIGERATION ANESTHESIA FOR AMPUTATIONS

The use of refrigeration as the complete anesthetic for a limb amputation is occasionally indicated. The person concerned is usually elderly, diabetic, and in poor general condition, coming from a slum district where his previous medical attention has been sketchy or nonexistent. His diabetes has been out of control and becomes more so in association with the severe infection of the foot and leg. He is semicomatose, irrational and running a high fever. Local examination reveals a blue, foul-smelling,

CHAPTER XXXIV

AMPUTATIONS

JOSEPHUS C. LUKE, M.D.

It is a distressing fact that, despite the recent advances in surgical diagnosis and treatment, the number of limbs requiring amputation is increasing rather than decreasing. The reasons for this are those of the changing modern life with its high degree of mechanization, its increased life span allowing for a greater frequency of the circulatory ailments of advancing age, and the fact of two destructive world wars in the first half of this century. Modern surgery has saved many lives in the amputated group, increasing the number of amputees, but has failed in most instances in devising means of resuscitating an ischemic limb. Therefore the need for amputation arises sufficiently often that every surgeon should be completely familiar with the indications and technique.

INDICATIONS FOR AMPUTATION

1 **Trauma.**—The only true indication for amputation following injury is irreparable damage to the blood supply to the limb and development of complete ischemia of the distal part. Such an injury is usually associated with major nerve damage and multiple fractures.

2 **Arterial Occlusion.**—As mentioned previously the most common indication in civilian life is occlusive arterial disease. The increasing age of the population and the prevalence of arteriosclerosis with or without diabetes necessitate the removal of many limbs because of gangrene. Buerger's disease, frostbite, acute arterial spasm, acute arterial thrombosis and embolism are less common conditions leading to the same result.

3 **Infection.**—It is rare in this era of antibiotics for a limb to be sacrificed because of

acute or chronic infection. The only exception is the case of widespread gas gangrene not localized to a single group of muscles, and it is not too much to hope that this type of infection will soon be controlled by new chemotherapeutic agents.

4 **Malignancy.**—Amputation of the limb well above the lesion is still the chief method of therapy in sarcoma involving the extremity.

5 **Deformity and Uselessness.**—Amputation for these reasons is becoming less and less practiced because of the improvement in orthopedic methods and the use of superior orthopedic appliances. This cause finds its greatest frequency in the removal of useless fingers and toes.

SELECTION OF LEVEL OF AMPUTATION

Where amputation is necessary following acute trauma, as much as possible of the limb should be saved. This means that amputation is carried out at the upper limit of the site of trauma. This should be done regardless of whether the stump is going to be final or require revision. This rule is even more important in battle than in civilian casualties, because, in the latter group, continuous uninterrupted treatment is possible. The reason for this selection of amputation level is that contamination or actual infection is always associated with open wounds and hence the original amputation level is unlikely to be a properly healed wound, especially if done by the guillotine method. An unsightly or unsuitable stump can always be revised at leisure when the initial wounds have soundly healed.

The same ruling applies in cases of frostbite where the gangrene is always more ex-

end weight-bearing can be obtained with almost the full length of the leg. However, a prosthesis must be worn for walking and it is of necessity an unsightly one with a bulbous ankle and lower leg corset, making it unsuitable for women. This amputation is absolutely contraindicated in occlusive arterial disease because of the poor blood supply to the long plantar flap and therefore it is used only in cases of trauma. During World War I, the greatest number of Syme's amputations were performed, but the majority of these had to be reamputated at the site of election because of painful or ulcerated stump. However, it should be said that many Syme's amputations have secured lasting excellent service, and it is claimed by the proponents of this type of amputation that the poor results are subsequent to improper selection of cases or incorrect surgical technique.

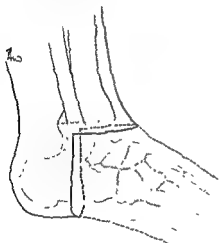


Fig 296—Syme's amputation

The tibia and fibula are divided just above the ankle joint through a transverse incision passing anterior to the ankle, and a long plantar flap including all the soft tissues of the heel is swung forward to cover the bone ends and to become the end-bearing surface.

Site of Election

This level is probably the most useful in all respects for a leg amputation. It allows

a functioning knee joint and the prosthesis is easily worn and gives a good cosmetic result. Unfortunately, it has a limited application in cases of arterial disease because of the attenuated blood supply below the knee, but the occasional successful result follows a careful preoperative evaluation as outlined in the paragraph on selection of amputation levels. It has its greatest usefulness in cases of severe foot trauma. However, the prosthesis must be carefully fitted and the skin over the tibial end must be movable to prevent the frequent complication of pressure sores over this area.

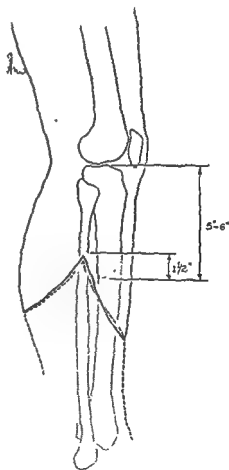


Fig 297—Site of election amputation

The length of the stump should be measured from the level of the insertion of the hamstring muscles on the tibia and fibula. Between five and seven inches of tibia is the ideal, but a useful stump can be obtained as short as two inches. Less than this makes

swollen, crepitant foot and leg, and it is obvious that this leg will result in his death from anaerobic infection if not soon removed. He will not stand a general or spinal anesthetic and time will not allow much in the way of expectant treatment.

The leg to the upper thigh is encased in chopped ice to which salt has been added and supportive intravenous and diabetic care commenced. After two hours a tourniquet is applied to the thigh just distal to the upper refrigeration level and the refrigeration is continued. One hour later painless low thigh amputation can be performed. Occasionally a slight spasm is felt by the patient when the sciatic nerve is severed, but otherwise the operation is painless and not shock producing.

The disadvantages to this form of anesthesia are the delay in wound healing due to prolonged tissue cooling and the increased incidence of wound infection, but it is undoubtedly lifesaving in the occasional indicated case.

SPECIFIC AMPUTATIONS

Toes

Toe amputations are indicated chiefly for deformity, trauma, or deficient circulation. Removal of part of the toe should be reserved for the first two indications mentioned, and is done, if possible, by a long plantar and short dorsal flap. Circulatory



Fig. 294—Amputation of toes

ailments demand removal of the complete toe; this is carried out by an elliptical incision with lateral flaps. The big toe is the most important one, being an essential factor in proper walking and balance and consequently every attempt should be made to conserve as much of this toe as possible.

Transmetatarsal Amputation

Transmetatarsal amputation is frequently used in severe crushing trauma to the forefoot and in those cases of peripheral arterial disease where preoperative tests in a case of painful or gangrenous toes indicate that healing will likely occur at this level. This amputation has supplanted the time-honored Lisfranc and Chopart types where, in the former, disarticulation of the tarsometatarsal



Fig. 295—Transmetatarsal amputation

joints was done and in the latter where the foot was removed by section through the midtarsal joint. In the transmetatarsal variety, a longer plantar than dorsal flap is fashioned if the blood supply to the foot is normal, or equal flaps are used when arterial disease is present. The metatarsals are sawn through as close to their bases as possible and all loose tendons are excised. The flaps include skin, subcutaneous tissue, muscle and fascia. The only disadvantages to this operation are the interference with balance of this foot and the tendency of the foot to inversion resulting from pull of the tibialis anticus attachment.

Syme's Amputation

The only advantage of Syme's amputation over a site of election removal is that

patella and the cut distal end of the femur in the Stokes-Gritti type sometimes fails to occur, and in all types, the stump end is bulbous due to the flare of the remaining portion of the condyles. This fact added to the longer thigh resulting from the artificial knee joint makes this type of amputation unpopular with the limb maker.

The two main varieties of end-bearing above-knee amputations are the Stokes-Gritti and the Callander types. In the former, the femur is divided at the upper level of the condyles and the articular surface of the patella is removed. A long anterior and a short posterior flap are necessary. The patella is retained in the anterior flap and the bare surface of the patella is fixed to the cut surface of the femur. Bony union between the two is a necessity. The Callander type is somewhat the same except that the patella is removed from the quadriceps expansion and the cut end of the femur is covered by this expansion.

Thigh Amputations

It is the opinion of most surgeons interested in peripheral vascular disease that when a good site of election amputation is not feasible, the lower third of the thigh is the next best choice. At this level the arterial supply is adequate, long flaps are unnecessary, and wound healing is thereby improved. A good functioning prosthesis can be easily fitted. The majority of amputations done for the complications of obliterative arterial disease will be done at this level.

Two and one-half inches above the knee joint is the optimum level, but an adequate stump is obtained from the middle of the middle third of the femur to this level. Equal flaps can be used, but a slightly longer anterior one is preferable. The subsequent shortening of the stronger hamstring muscles pulls the suture line posteriorly even when the flaps are initially equal. Suturing of the cut muscle over the bone end is not advisable when it is likely that a prosthesis will be worn because the resulting bulky end

of the stump makes for difficulty in fitting a proper socket. However, in patients who are unlikely to wear an artificial limb, this point has advantages in cushioning the stump end. The author prefers to cut the muscles at the level of bone section and retain only the deep fascia with the skin flaps. The nerves are cut as high as possible to keep them clear of the scar. The sciatic is the only one which should be ligated at the time of division because of the artery which runs within.



Fig. 301.—Supracondylar amputation.

The least amount of thigh possible necessary to guide a prosthesis is six inches from the great trochanter, consequently every effort should be made to achieve this length. If a higher amputation is necessary because of trauma or malignant disease, the head and neck of the femur should be retained to preserve hip contour. This is more desirable and is easier than hip disarticulation.

for great difficulty in having the below-knee section activate the prosthesis. When the amputation level is less than three inches of tibia, it is preferable to excise the entire fibular fragment. This prevents springing of the fibula away from the tibia and the more successful achievement of a conical stump. In traumatic cases a longer anterior than posterior flap is cut, but in amputations due to arterial disease, equal flaps are the rule. These flaps, as in all amputations, should be slightly larger than the diameter of the limb.



Fig. 298—Recent below-knee amputation because of gangrene of the foot due to Buerger's disease

End-Bearing Amputations at the Knee

With the improvement in the construction of artificial limbs, the necessity of end-bearing stumps for proper walking is becoming markedly diminished. Although eminently satisfactory as a functioning stump, this type

of amputation has several disadvantages. The long anterior and short posterior flaps make for circulatory troubles in the longer flap resulting occasionally in sloughing of the edges and consequently delayed healing. Bony union between the cut surface of the

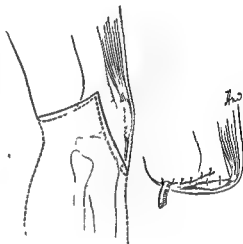


Fig. 299—Stokes-Gritti amputation



Fig. 300—Previous left mid thigh amputation under refrigeration anesthesia and recent right below-knee amputation in a case of diabetic gangrene

The level of removal of a damaged digit is open to considerable controversy, and it should be emphasized that the length of a finger should not be sacrificed to obtain skin flaps because such skin deficiencies can be easily remedied by skin grafting. In general the following statements are widely accepted.

The Thumb

The thumb is by far the most important digit, but it requires parts of the other digits for proper opposition. Therefore, the most conservative attitude is required in treatment of its injuries; all possible length is preserved using skin grafts. All possible length of the other damaged fingers is also indicated for the best possible opposition.

The index finger is next in importance. Traumatic amputation of the tip is best treated by skin grafting to a pedicled graft lifted from the skin of the thenar eminence; or by a split thickness skin graft. If more than the distal one and a half phalanges are destroyed, amputation is best through the metacarpal to give optimum cosmetic appearances. This latter point applies only when the thumb is undamaged, and should be considered only as a secondary procedure in the laborer.

The middle finger can be amputated at any point because any length of stump adds strength to the hand. Therefore, in the manual worker, conserve as much as possible of all phalanges. In the white collar worker who has lost the majority of this finger, it is probably wise to remove the entire finger and the complete metacarpal. This allows the index finger to take over the function of the middle and gives a much better cosmetic result.

The ring finger can be treated on much the same lines as the middle.

The little finger is of least importance. Amputations are successful at any level, but when most of the finger is destroyed, the head and most of the shaft of the meta-

carpal should be removed to give best cosmetic results. The metacarpal head is best preserved in the laborer.

Forearm Amputations

Forearm amputations are indicated mostly as a result of severe trauma. Occlusive arterial disease rarely involves the upper extremity to the point of needing amputation. Weight-bearing and strain on the stump in the prosthesis are not factors to be reckoned with, consequently upper extremity amputations rarely are heir to later complications. Equal flaps are therefore indicated. The optimum levels are not more than 7 inches from the olecranon to a minimum length of 1 inch beyond the insertion of the biceps tendon. Less than this amount leaves insufficient length for the forearm stump to control the artificial elbow. A disarticulation at the wrist has little to recommend it and is inferior to a 7-inch forearm stump.

Upper Arm Amputations

The same observations as in the forearm apply to the upper arm. The optimum level is eight inches from the acromion process and the minimum functioning stump is one inch from the anterior fold of the axilla.

Fore Quarter Amputation (Interscapulothoracic)

The indications for this procedure are the same as in the hind quarter removal. The arm including the scapula and all or most

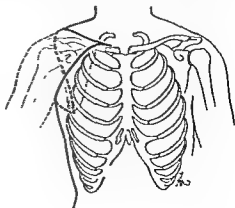


Fig. 303.—Forequarter amputation.

Hind Quarter Amputation (Interinnomino-Abdominal)

This heroic amputation is indicated only in primary malignancies involving the head and neck of the femur or the pelvic bones on one side. It consists of removal of the limb and half the pelvic girdle, after division of the symphysis pubis and ligation of the common iliac vessels on that side. A portion of the sacrum may need removal if the position of the tumor so demands. The cure rate is understandably low when the operation is done for osteogenic sarcoma but is better in chondrosarcoma and fibrosarcoma.

UPPER EXTREMITY AMPUTATIONS

Fingers

Whether a finger should be amputated after trauma of a crushing type depends upon many individual factors. No hard and fast rule can be laid down, but, in general, it should be said that if there is any possibility of obtaining a moderately functioning finger, then conservatism is indicated. A poor result can always have a subsequent amputation. Because of the frequency of poor results, most large hospitals have organized a Hand Group, who, with their teamwork, especially between the physiotherapist, traumatic and plastic surgeons, achieve better results than those usually obtained. It is in the segregation of these cases under the care of such a well-trained group that the hope of improvement in results rests. In general, it can be said that amputation is indicated when, due to injury, pain or sepsis, the finger becomes a menace to the individual, when it is useless or actively in the way, or where it interferes with the use of the hand.

Other points to be kept in mind are:

1. The workman requires a strong mobile hand. Stiff fingers interfere with his manual labor and must be avoided. The cosmetic factor is of secondary importance. Therefore, in general, immediate finger am-

putation is more indicated in trauma than prolonged plastic procedures.

2. In children, the most conservative approach is taken because of their wonderful powers of functional restoration.

3. In men and women of the executive type, the cosmetic factor is most important and great strength is a secondary consideration.

4. It is never necessary to suture tendons over the amputated ends of fingers because this interferes with the function of the other digits.

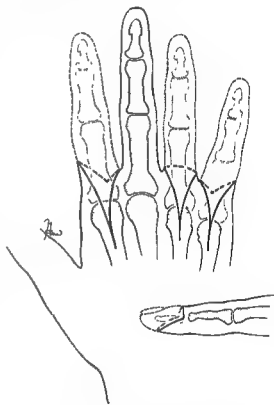


Fig 332—Amputation of fingers

5. When amputation takes place through joints, the cartilage should be removed to prevent a bulbous and sensitive stump. Cartilage is also more subject to infection, fistula and sequestration than bone.

6. If amputation is in the region of the nail bed, the nail matrix should be totally removed to prevent the development of horny nail growths.

artificial limb should be applied as soon as possible, but in most cases the stump is not adequately shaped before two months. The wearing of temporary "peg legs" or pylons is not advisable because they lead to incorrect walking habits. Later corrections to the initial socket are always necessary as continued stump shrinkage occurs. Good fit, lightness, durability, and good repair service from the limb maker are essential points in an artificial limb. The most recent advance in limb fitting is the further development of the original German idea of the suction socket for thigh amputation. In these the stump is held in a close-fitting bucket in which a negative pressure is produced by exhausting the air in the socket through a distal valve when the stump is inserted. This

allows more natural walking and dispenses with the pelvic band necessary in the conventional types. Cases suitable for this design must, however, be carefully chosen and must not be those done for occlusive arterial disease.

REFERENCES

- Callander, C. L.: New Amputation in Lower Third of Thigh, *J. A. M. A.* 105: 1746-1753, 1935.
Luke, J. C.: Amputation for Ischaemic Arterial Disease of the Leg, *Canad. M. A. J.* 65: 343-347, 1951.
McKittrick, L. S.: Diabetic Gangrene; Review of 972 Cases, *Arch. Surg.* 40: 352-363, 1940.
McMaster, P. E., and Mazet, R., Jr.: Suction Socket for Above-Knee Amputees, *Surg., Gynec. & Obst.* 89: 335-338, 1949.
Pearl, F.: A Traumatic Low Thigh Amputation, *Surg., Gynec. & Obst.* 87: 308-312, 1948.
Richards, V.: Refrigeration Anesthesia in Surgery, *Ann Surg.* 119: 178-200, 1944.

of the clavicle are removed along with the associated muscles, axillary fat, and glands. The subclavian vessels must be tied early in the operation and the brachial plexus divided.

AFTER-CARE OF AMPUTATIONS

It is the surgeon's responsibility not only to do the amputation and assure a good convalescence, but also to be certain that the patient is physically and mentally prepared for his artificial limb. Loss of a limb is a great psychic shock to any individual, and the ability of the patient to adapt himself to his artificial limb will depend on his mental attitude toward it. The surgeon can do a great deal in promoting a healthy mental attitude of his patient toward his disability. Early postoperative mobility, occupational therapy, moving pictures of the activities of other amputees and personal visits to the patient by other adapted individuals with loss of a limb will be a big boost to morale. Certain patients, however, never are capable of wearing a prosthesis, and expensive and misguided efforts in obtaining a limb for such a person should be discouraged. Many persons in the sixth, seventh or eighth decade never acquire the balance or will power to use crutches successfully, let alone learn to use an artificial leg. Such patients have usually been bedridden for months before amputation is finally done. Their musculature has wasted and their morale is poor. In most of these patients the remaining limb is also involved by arterial disease, and in about 30% its amputation is necessary within three years from the initial one.

Despite the possibility that an artificial limb may never be worn, all amputations should be treated postoperatively as though that possibility did not exist. The cardinal principles of aftercare of the stump are:

1. Shaping of the stump by bandaging to control edema and prepare the limb for the prosthesis.

2. Exercise of the stump to enable the amputee to use the artificial limb adequately.

3. Prevention of stump contractures

CINEPLASTIC AMPUTATIONS

The plan of employing individual muscles or groups of muscles to activate a functioning part of the prosthesis was first conceived by Vanghetti in Italy at the close of the last century. A slow development of this technique has taken place, but has not been widely accepted because of the inability of the limb maker to produce an artificial limb capable of properly utilizing this surgical advance. However, since the close of World War II, renewed interest has been aroused by the development of improved prostheses, and it is likely that the usefulness of this method will be more appreciated and more widely used.

The upper limb only is suitable for this modification because the finer movements achieved in an artificial hand are unnecessary in the leg. In general, the technique consists of utilization of a muscle or agonistic group of muscles to act on a skin tunnel around which the appropriate tendons have been fixed. This action is transmitted to a rod in the skin tunnel which is connected to activate the artificial fingers (thumb and index). Powerful and delicate voluntary movements can be achieved allowing for the grasp of any object from that of a match to a hammer. The formation of the so-called "motor" loop can be accomplished at the time of the initial amputation when infection is not a possibility or following the initial amputation. However, if done later, it must be ascertained that the individual muscle has sufficient length and still is an active contractile unit. Intensive training and use of the utilized muscles is a most important postoperative measure.

THE PROSTHESIS

No two amputations are of exact similarity even though of the same type, hence each limb has to be made to order. The correct

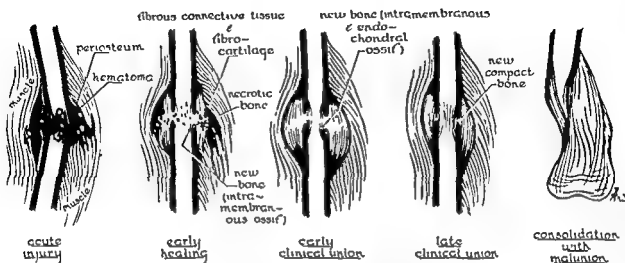


Fig 304 —Stages in the healing of fractures (after Urist).



Fig 305 —X-ray of clinical union.

CHAPTER XXXV

FRACTURES

H F. MOSELEY, D M

A fracture may be defined as a breach in the continuity of a bone which is complete or incomplete. Fractures are of many types depending on the mode of classification, thus

A Traumatic—when due to an adequate force of

- 1 Direct violence
- 2 Indirect violence.
- 3 Muscular violence.

B. Pathological—when the fracture is largely due to disease in the bone caused by

- 1 Congenital defects; fragilitas ossium, pseudarthrosis of tibia
- 2 Metabolic errors; rickets
- 3 Generalized bone disease; Paget's disease, hyperparathyroidism
4. Generalized osteoporosis, old age, invalidism
5. Local inflammation, osteomyelitis
- 6 Neoplasm, benign or malignant

Fractures may be classified according to the absence or presence of a communicating skin wound into

- 1 Closed (simple).
- 2 Open (compound)

The use of the word *simple* is probably misleading and should be replaced by the word *closed*. Very few fractures are simple to understand or treat. It is well to realize that a fractured tibia is best regarded as a patient with a fractured leg and the problem must be so assessed and treated. Too much emphasis must not be placed on the bone alone; adequate attention must be given to the soft tissues of the affected part, and the patient as a whole in relation to his work and family.

An open or compound fracture is one in which a wound in the skin and soft parts

places the environment in communication with the fracture site.

There are two types of open fracture:

- 1 Direct—in which the direct violence compounds the fracture from without.
- 2 Indirect—in which the indirect violence causes the fractured fragments to compound the soft tissues from within.

Birth fractures occur in relation to the mechanics of parturition and may involve any of the long bones of the upper or lower limbs.

Greenstick fractures occur in children before puberty when the bones are elastic like green twigs and bend rather than break. When the fracture separates at the epiphyseal plate with or without a fragment of metaphysis, the injury is called an epiphyseal separation.

A sprain fracture results from a joint displacement where the ligament avulses its bony attachment in place of rupturing its fibers

Fractures may be closed or open, complete or incomplete, or complicated by associated lesions of muscle, nerve or blood vessels

REPAIR OF FRACTURES

The process of repair follows immediately the occurrence of fracture. A hematoma develops around the broken fragments and injured soft tissues. Traumatic inflammation ensues with hyperemia, edema, resultant swelling, heat, local pain, and tenderness.

The fracture hematoma is widely considered the basis on which the healing process develops. The hematoma is replaced by granulation tissue from which callus, the healing cement of bone, arises. Into this callus, calcium and phosphate ions are pre-

of severe internal injuries in the transport accidents of this age.

In fractures involving the limbs, a careful study of the circulatory and neurological conditions must be made. This is especially true in open fractures and fractures associated with marked displacement or dislocation.

In evaluating the clinical picture, the following factors must be considered:

1. **History of Accident.**—A detailed account of the accident must be obtained. The exact hour, the method of handling at the time, the manner of transport to hospital, time of arrival in the ward and operating room are significant from a medical and possibly a legal point of view. The severity of the injury gives an approximation as to what may be expected in bodily damage.

2. **Local Symptoms and Signs of Fracture.**—

(a) **Pain**—exquisite pain is felt in the region of the fracture. The muscles are in spasm and any movement augments discomfort.

(b) **Tenderness** is noted on palpation of the fracture site.

(c) **Swelling** may be present from extravasation of blood, or from displacement of the bone ends.

(d) **Deformity** occurs in the presence of angulation or overlap of the bone fragments.

(e) **Excessive mobility** and bony crepitus will be noted on examination of a complete fracture.

(f) **Loss of function**

(g) **X-ray examination** is imperative and gives information regarding the exact injuries and displacement of the bony skeleton. It must always, however, be considered as an accessory to accurate clinical observation of the patient although elicitation of crepitus and painful examination are rendered unnecessary.

EMERGENCY TREATMENT

General.—When first seen, the patient should be given mental reassurance; seda-

tives are administered to allay pain, and diminish psychic trauma. If operation is contemplated, preparations are arranged accordingly.

Local.—The part should be splinted securely and all unnecessary movements avoided. The affected limb should be elevated and an ice bag applied.

If the fracture is open, apply a sterile dressing. When hemorrhage is present, apply a pressure dressing and elevate the limb. A tourniquet is rarely necessary. If used, it should be released every 15 minutes and only pressure, adequate to obliterate the pulse, applied.

Antitetanic serum should be given in all open fractures. The patient should be prepared for the operating room at the earliest moment.

ORGANIZATION OF FRACTURE TREATMENT

An organized approach is required to secure uniformly good results in fracture work. The principles of this organization are as follows:

(a) **Segregation of cases.**

(b) **Uniformity of treatment** with standardization of methods and equipment.

(c) **Supervision** by the same personnel from the initial to final treatment.

(d) **In all cases** a planned program for each patient. This should include physiotherapy, occupational therapy, and general rehabilitation.

Fundamentals of Fracture Treatment

The ideal of fracture treatment is to secure the maximum return of function in the minimum time.

(a) **Reduction.**—Displacements are reduced to their anatomical position if possible.

(b) **Immobilization.**—The fracture is immobilized by splinting until united.

(c) **Protection.**—The part is protected during the period between clinical union and consolidation with graduated return to full function.

cupitated from the blood and from mobilized minerals of the bone ends. The two forces directing the course of repair are the vital activities of the osteoblasts in the early and late stages and after a certain point functional stresses and strains. The circulatory status of the healing tissues is very important.

The general method followed by Nature is to lay down first a *temporary* scaffold on the periosteal and endosteal aspects, which permits the *permanent* or *definitive* bone to be laid down later in orderly fashion between the compact surfaces

necessary as a permanent buttress. X-ray, however, at this stage will not show bony trabeculae crossing the fracture line, and the line will still be visible.

Consolidation or final union requires a much longer period and is present when the x-ray shows the restoration of normal architecture with loss of the fracture line and trabeculation across this area completed. Consolidation or final union is therefore today an x-ray interpretation. Function may be possible for months before this stage is finally reached in fractures of the long bones



Fig. 306—X-rays showing consolidation of fractured tibia sustained 6 years previously and also a fresh fracture dislocation of the ankle

Clinical union may be said to be present when the fractured bone will move as a unit without pain or local tenderness. At this stage, guarded and graduated stresses and strains will accelerate the further consolidation of the healing by laying down the final architecture according to Wolff's law. As the permanent callus gains strength, the temporary callus absorbs except where

CLINICAL PICTURE

Patients with fractures present a variable picture, from the minor complaints that might suggest merely a sprain or bruise of bone to the severe symptoms of shock seen in open, complicated, or multiple fractures. All require a careful general as well as local examination, bearing in mind the possibility

is often best delayed until the swelling is lessened by elevation of the part. Should, however, marked displacement, dislocations, or complications to blood vessel or nerve be present, immediate reduction is indicated.

and attached to weights through stirrups, cords, and pulleys, i.e., *skeletal traction*.

The countertraction in these cases is usually the body weight. As in these cases the patient is free to move the body and affected limb, this form of continuous traction is called *mobile traction* in contrast to *fixed traction* as is employed when the Thomas splint is used for emergency transport of a fractured femur.

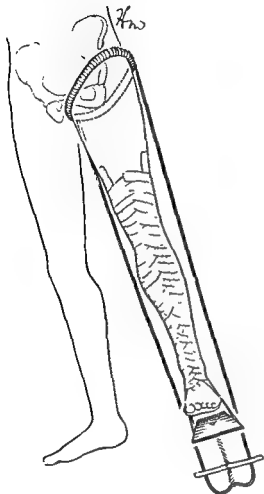


Fig. 307.—Fixed traction on Thomas splint.

Reduction by Traction.—In fractures, such as oblique fractures of the femur, reduction may be obtained and maintained by the use of continuous traction. This is of greatest value in cases such as the above-mentioned where the fractures once reduced are unstable because of the nature of the bony surfaces and the muscular forces acting upon them

Continuous traction may be applied through adhesive straps applied to the skin, i.e., *skin traction*, or by the use of Kirschner wires or Steinmann pins transfixing the bones

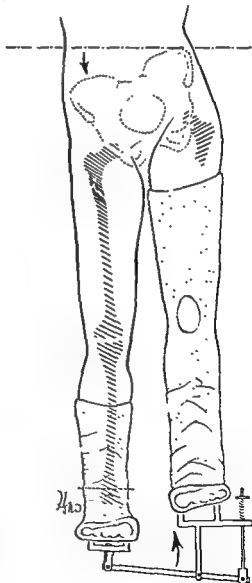


Fig. 308 —Well leg traction.

When the lower limb is arranged in mobile fashion with the Thomas splint used as a cradle and the traction on the limb by weights over pulleys balanced by elevation

(d) **Functional Treatment of Soft Parts.**—From the earliest moment, function of the soft parts is encouraged. The patient is made ambulatory at the first opportunity. Return to light work precedes full duty, and rehabilitation should be an essential part of the treatment.

REDUCTION

Reduction means the restoration of the displaced fragments to as near the anatomical position as possible. Perfect reduction is the ideal, but this cannot always be achieved because of the nature of the injured surfaces, the interposition of soft parts, and the later influence of altered muscle pull, splinting, and gravity. For this reason a knowledge of what constitutes adequate reduction is of the greatest importance.

The age of the patient determines the plasticity of the bone. In infants and young children, the healing power of bone is very great and often fractures uniting in positions of gross displacement will finally in the course of time be reformed to the shape of the normal bone in the area. This quality of bone diminishes rapidly from birth to old age but at any age this fact should not influence our desire for perfect reduction.

The Site of Fracture.—Fractures in the shafts of the long bones of the *upper limbs* may heal with overlap and sometimes with angulation without gross interference to function of the limb as a whole. One joint may be disorganized by malunion but the other joints usually increase their range of function to compensate for this defect. Shortening of the limb by one or more inches is not usually obvious. In the *lower limb*, however, shortening of any degree over one-half inch results in inequality of leg length and requires compensation by correction to the shoe. Without this a limp may be present. Any alteration of the normal direction of force through the joints due to angulation is followed by abnormal stresses and strains which, because of the transmission of body

weight, leads to wear and tear changes or traumatic arthritis.

If the site of fracture involves the *joint surfaces*, exact anatomical reduction is required, otherwise the smooth gliding action is lost and, as is found in machinery, rapid deterioration of the mechanism results. Joint injuries frequently require removal of loose fragments of bone in order to prevent the rapid development of traumatic arthritis.

It follows that reduction necessitates the restoration of the parts to their normal position, reestablishing the proper length of the bone and therefore of the limb, and with this the correct transmission of forces through the bone, joints and limb as a whole. Angulation of the fragments is the chief defect to be avoided, and it is preferable to have overlap than angulation—since in overlap the forces through the joints above and below approximate the normal.

Methods of Reduction

Most methods of reduction employ the use of traction applied to the distal and counter-traction applied to the proximal fragment. Direct pressure may be required to the area of fracture to correct angulation or lateral displacement. Such manual operations are called *manipulative reductions*. Reduction is assessed by the gross appearance, palpation, and x-ray examination.

Manipulative Reduction.—Success with this method is determined by the use of.

- 1 Knowledge of the anatomy and physiology of the musculoskeletal system

- 2 The relaxed muscle position

- 3 Anesthesia to secure relaxation and freedom from pain, (a) local, (b) regional, (c) spinal, (d) general.

- 4 Fluoroscopic and x-ray examinations. Such records are of the utmost importance in medicolegal cases.

- 5 Correct timing of the operation. Manipulative reduction is best carried out at the earliest moment. Once gross swelling has occurred, reduction is more difficult and

of the foot of the bed, the expression *balanced traction* is employed.

Sometimes traction is applied to the sound lower limb, the counterpressure being the sole of the affected limb. This constitutes the principle of *well leg traction* employed occasionally for fractures of the upper end of the femur.

The most common use for continuous traction is in displaced fractures of the thigh or leg. Either the Thomas or Braun splint is used.

Distraction and Impaction.—A variation of traction and countertraction methods where complicated apparatus is involved are the methods of mechanical distraction followed by impaction with the use of external fixation for immobilization. The two sets of apparatus most commonly used are those of Roger Anderson and Stader. This method requires considerable experience for successful use and has not gained great popularity because of this difficulty and the danger of infected pin holes and joint stiffness.

Operative Reduction.—Operative reduction and the associated internal fixation of fractures were introduced by Lambotte and Lane at the beginning of the century. They were dissatisfied with the clinical results obtained in oblique fractures of the femur and tibia in adult patients. The method was correct but its application until recent years resulted in serious complications due to infection, poor surgical technique and foreign body reaction.

The present use of this method has been increased by the following considerations:

(a) Improved standards of technique and asepsis on the part of the surgeon, his assistants, and associated operating room personnel.

(b) The prophylactic and curative power of the chemotherapeutic and antibiotic drugs.

(c) The development of inert metal for plates and screws.

It is not the method for all fractures and should be used only in selected cases. Its

chief danger is still the fact that it converts the closed into an open fracture and invites disaster if complications ensue.

Indications.—

1. When manipulation will fail or has failed because of

(a) Interposed soft parts.

(b) Separation of fragments by muscular contraction—patella—olecranon.

(c) Delayed treatment with malunion.

(d) Certain intra-articular fractures.

2. When reduction cannot be maintained. Oblique fractures of femur, tibia or femoral neck.

3. In fractures complicated by injury to blood vessels, or nerves, necessitating their exposure.

The guiding principle in the reduction of fractures is to employ the correct method, for the correct case, in the correct manner, at the correct time, and to select when possible the nonoperative procedures.

IMMOBILIZATION

Plaster of Paris Technique

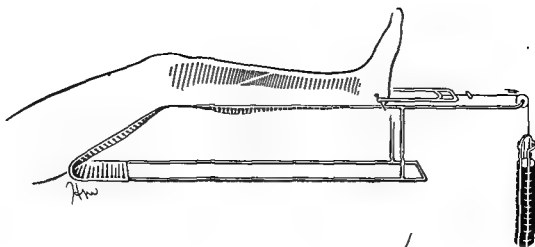
This constitutes the most useful of the methods for the immobilization of fractures. Since the introduction of the plaster bandage by Mathijsen in 1852, the technique has greatly improved, and today casts are applied in such a way as to preserve the maximum function of the part and to permit early ambulation. The most important principle is to apply the lightest possible splint with the parts held in the positions of optimum function. Excessive weight of cast is to be avoided since the requirement is a patient with an attached splint, not a splint with an attached patient.

Method.—The plaster bandage and slab are the building blocks of the finished cast, and in order to standardize the work it is necessary to have uniform materials.

Uniform bandages and slabs must possess:

(a) A standard width and length.

(b) A uniform amount of plaster applied to crinoline or mesh.



sites of
application
of
skeletal
traction—

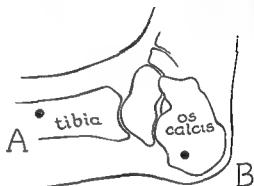


Fig 309—Skeletal traction on Braun splint

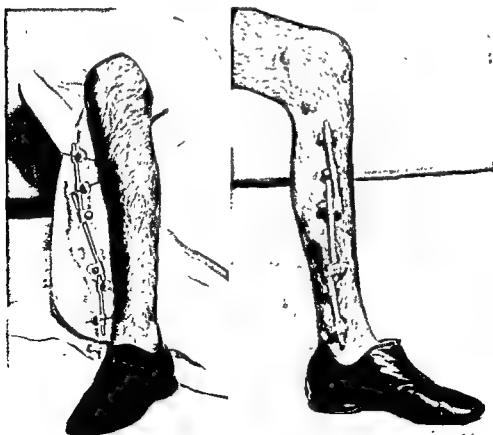


Fig 310—External skeletal fixation for fractured tibia.

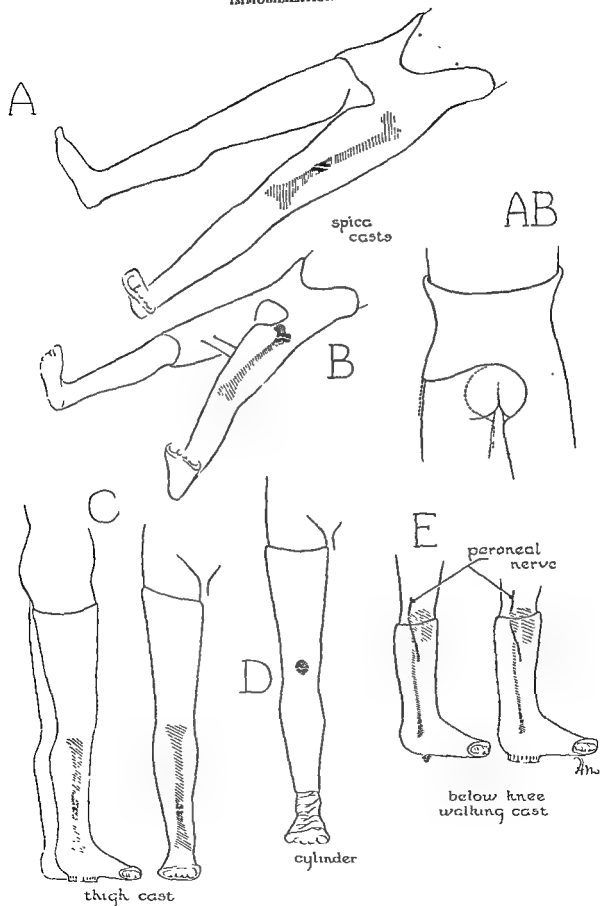


Fig 312.—Type casts for fractures of the lower extremity.

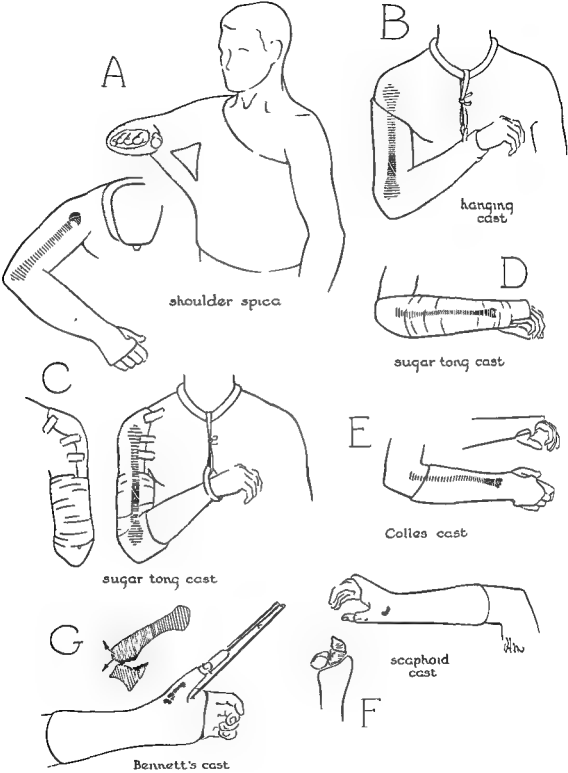


Fig 311 —Type casts for fractures of the upper extremity

the neck of the fibula. This may be damaged by the upper edge of a below-knee cast or by pressure from an unpadded area of the hip spica or thigh cast.

4. *Loss of reduction* may result if the cast is applied with too much padding or when the limb is swollen. In both cases the cast loosens in time. This is obviated by avoidance of excessive padding and change of cast on subsidence of edema.

Traction.—In cases in which traction has been used to secure reduction, immobilization may be obtained by the use of continued traction. This is especially useful in fractures of the shaft of the femur.

External Fixation.—The use of two pin units and connecting bar can be employed for certain cases.

Internal Fixation.—This may be accomplished by the externally applied plate, the Smith Petersen pin, longitudinally placed Kirschner wires, or by the use of the intramedullary nail (Küntscher). Immobilization by internal fixation is usually supplemented by a plaster cast until clinical union is obtained.

PROTECTION UNTIL CONSOLIDATION

A considerable interval of time intervenes between clinical union and final consolidation as demonstrated by x-ray examination. In the upper limb, this protection can be secured by graduating the function of the part and avoiding sudden strain or injury. No special apparatus is required.

In the lower limb, the protection is secured by graduating weight-bearing through the use of crutches and cane. In fractures of the femur, the Thomas walking caliper is an additional support. In fractures of tibia and fibula the caliper or a plaster cylinder may be worn

FUNCTIONAL USE OF THE SOFT PARTS

The earliest moment after the initial treatment, the patient is informed of the extent of his problem and the injury assessed in

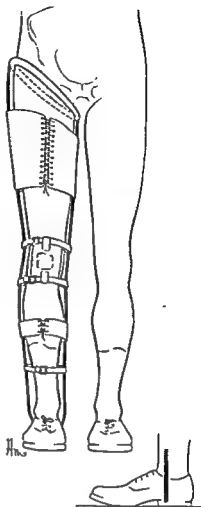


Fig. 313.—Walking caliper (Thomas).

relation to his possible time in hospital, absence from home and work, and the economic factors involved. His cooperation both mentally and physically is required for efficient treatment, and this can be secured only if he understands the manner in which he can assist the surgeon. The doctor's main thought must be directed toward the problem of the patient as a whole in relation to his environment, and he must try to fit the treatment of the fractured limb to this pattern.

The greatest benefit is usually rendered by assuring the patient that he will eventually make a good recovery and return to full work; that the period required will be considerably shortened if he will actively contract all muscles of the part even though covered by a plaster cast; that active movements of all unsplinted joints and the con-

(c) A standard time for hardening

(d) A minimum of shrinkage during the process of drying.

The plaster may be applied directly to the skin. This is called the *nonpadded* technique popularized by Bohler. Most workers cover the limb with stockinette and place a variable amount of sheet wadding over bony prominences before the plaster is applied. This is the more popular method and is called the *padded technique*. The danger of pressure effects on the skin, circulatory mechanism or superficial nerves of the part is thereby lessened.

The bandage is immersed in water until the bubbling ceases and is then applied evenly without constriction taking care to smooth one layer against the other and remove all air spaces. After the cast is applied, hardening may be accelerated by the use of an electric drier or baker.

If the cast is applied to a fresh fracture and swelling is expected, the circular cast is split to enable this to occur without danger to the limb. Elevation of the limb is essential. *The patient is taught to contract all muscles possible under the cast and to move all adjacent unsplinted joints.* In the upper limb active use of the fingers is most important. The splinted part is carefully observed for 36 to 48 hours following the application of the cast to see that no complications occur, such as interference with circulation.

Standard Casts.—

Upper Limb

Shoulder spica: For avulsion fractures of greater tuberosity.

For ruptures of rotator cuff.

Hanging cast: For fractures of shaft of humerus.

For fractures around elbow.

Sugar tong cast. For fractures of shaft of humerus

Colles' cast: For fractures of lower end of radius.

Sugar tong cast: For fractures of radius and ulna

Scaphoid cast: For fractures of scaphoid.

Bennett's splint: Traction with cast for Bennett's fracture dislocation

Lower Limb

Plaster spica: For fractures of hip or femur.

Thigh cast: For fractures of tibia and fibula

Cylinder: For fractures of patella

Below knee cast. For ankle fractures

Spine

Cervical collar. For cervical injuries.

Body jacket. For lower dorsal and lumbar fractures

Complications.—

1. *Plaster sores* result from continued pressure on the cutaneous tissues overlying bony prominences. The sacral area, anterior iliac crest, malleoli, heel, and bony points around the elbow are the sites most frequently involved. The thin, emaciated patient confined to bed is subject to these sequelae. The lower edge of the cast may cause ulceration of the skin if the limb distal to this point is swollen from dependency. Creases on the deep aspect of the cast from movements during the application may cause subsequent skin ulceration. This is especially frequent over the anterior aspect of the ankle.

2. *Circulatory disorders* are prone to occur if the cast is too tight. This may follow immobilization of the recently reduced fracture with subsequent swelling of the limb. Careful attention must be given to the peripheral pulse and cutaneous circulation. Pain and swelling necessitate relief of the constriction by splitting the cast and elevating the part. The cast may predispose to Volkmann's ischemic contracture in elbow injuries or even to gangrene of the distal part where the circulation is completely occluded for too long a period. Sympathetic block is an accessory therapeutic measure to be considered in such cases.

3. *Neurological lesions* may occur from direct pressure on nerve trunks. The most commonly involved nerve is the peroneal on

5. Infections near joints, e.g., skeletal traction pin sinuses.
6. Passive stretchings.
7. Disuse atrophy.
8. Reflex autonomic imbalance.

Treatment.—

Prophylactic.—

- (a) Maintenance of functional activity of all unsplinted joints and active contractions of all muscles under casts.
- (b) Avoidance of splinting in positions other than those of function.
- (c) Use of traction through joints of minimal required force, and only for periods absolutely necessary
- (d) Elastic support to prevent edema.
- (e) Use of sympathetic block to overcome autonomic imbalance.

Curative.—When stiffness has developed, the appropriate use of heat, massage and

active exercises is the basis of treatment. The range of movement must be measured and the progress charted to ascertain whether the therapy is beneficial. Forced manipulations and passive stretchings are best avoided.

DELAYED UNION AND NONUNION OF FRACTURES

The time required for the union of a fracture depends on many factors. It is therefore most difficult to define accurately when such healing is too slow.

Delayed union may be said to occur when the average time for the particular fracture has been passed and the x-ray shows decalcification and widening of the fracture line.

Nonunion is present when there is movement at the fracture line when tested clinically and the x-ray shows sclerosis of the bone ends.



Fig 314—Nonunion and malposition of tibia

stant use of the unsplinted fingers are essential

Encouragement is given by the physiotherapist who supervises these activities and employs heat, massage, contrast and whirlpool baths together with active exercises in his regime

In cases requiring prolonged institutional care, occupational therapy will keep the mind occupied and stimulate progress.

At the earliest moment, the patient should be made ambulatory and functional use of the part encouraged. Return to light duties should precede heavy routine work. In cases with Colles' fractures properly splinted, typing, driving a car, and such activities are permitted

In lower limb fractures, most sedentary occupations can be carried out in a walking cast. In severe injuries in the working class, a period in rehabilitation centers should precede return to full duty.

OPEN FRACTURES

In these cases, the principle is to treat the wound in the soft tissues so as to obviate infection and convert the open into a closed fracture.

This ideal may be possible where the soft tissue damage is not extensive and when the case is seen within 6 to 12 hours. In civilian practice with the use of chemotherapy and antibiotics the percentage of successful closures of such wounds is much higher than in the period preceding 1940. Extensive wounds in military practice are best left open and treated by secondary suture.

The initial treatment requires the general care of the patient and measures directed to combat hemorrhage and shock. Following this, a meticulous cleansing of the wound by excision of devitalized skin, necrotic tissue, fascia and muscle is performed in the operating room under appropriate anesthesia.

Displaced fractures are reduced and immobilization arranged by internal fixation or by external splinting. The wound is closed if this is possible without tension, otherwise

it is lined and lightly packed with fine mesh gauze. Local antibiotics may be placed in the wound. After 5 to 7 days the pack is removed and the wound is closed by secondary suture or plastic procedure.

Antibiotics are given systemically for at least six days. Antitetanic serum is administered routinely.*

COMPLICATIONS OF FRACTURES

Joint Stiffness

Restricted motion of joints immobilized in the treatment of fractures is a common complication. This stiffness varies from a minimal degree which is quickly lost by functional activity, to a more severe involvement, which may take months or even years before full range of movement is regained. The joints may remain permanently disabled.

The underlying cause for the joint changes is the development of adhesions which fix the synovial plications and the fascial planes between muscles, ligaments, and capsular structures. With these alterations there is frequently a lessening of synovial secretion caused by disuse. The muscles atrophy and the joint assumes a characteristic deformity determined by altered muscle balance and gravity. In time absorption of cartilage causes a diminution of joint spacing

Factors Predisposing to Joint Stiffness

1. Interference with circulation to the distal part of the limb caused by splinting in a faulty position. For example, splinting cases of Colles' fracture in forced volar flexion in the older patient results in swelling of the hand and fingers with subsequent stiffness
2. Excessive traction through joints such as the knee or fingers
3. Reactionary edema after injury to a limb
4. Effusions into joints often result from ligamentous damage.

*Gas Gangrene (See section on Surgical Bacteriology and Chemotherapy)

achieved without shearing at the fracture line.

7. Old age, invalidism, poor nutritional state.

8. Pathological conditions of bone. When the fracture occurs through a malignant growth of primary or secondary nature.

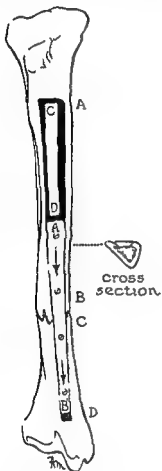


Fig 316—Sliding bone graft of tibia

Degrees of Nonunion —

1. Absolute nonunion—a gap exists, e.g., separated fracture of patella.

2. Fibrous union—ends are sclerosed and dense fibrous tissue exists between the fragments.

3. Pseudarthrosis—a false joint has developed.

Treatment.—Delayed union and nonunion are best prevented by correctly applying the proper treatment to the fresh fracture. When delayed union is diagnosed the first

consideration is given to the probable causes which have been enumerated, and if possible such are corrected. Often proper immobilization of the fracture associated with graduated function will accelerate the healing process.

When sclerosis of the bone ends has occurred and nonunion is established, the use of a bone graft to open up fresh vascular channels in each fragment, and at the same time afford internal fixation, is the procedure of choice. Drilling of the sclerosed ends after exposure or freshening the bone ends and application of a metallic plate may be used in certain cases.

Malunion

When fractured fragments have united in an overlapped, angulated or displaced position, the fracture is said to be *malunited*.

Causes.—

1. Failure of adequate reduction.
2. Failure of adequate immobilization.
3. Displacing forces of muscular pull, faulty traction or faulty splinting.

Treatment.—If sufficiently severe to interfere with adequate function or for cosmetic reasons, the area may be exposed, the bone refractured, and the corrected position obtained.

Immobilization is then secured by internal or external fixation.

Myositis Ossificans

When muscle, ligament or periosteum is avulsed from its bony attachment, a hematoma develops. In this matrix osteoblasts proliferate and produce bone. Irritation during the early stages by stress or strain increases this bone formation. The most common site for this development is around the brachialis anticus insertion after dislocation of the elbow.

Treatment.—When the presence of the calcified shadow is seen on x-rays after a joint injury, attention should be directed to prevent any passive stretching of the area. No operative procedures should be consid-

Factors Which Tend to Delayed Union and Nonunion.—

1 Inadequate reduction. In certain fractures such as those of the patella or olecranon the fragments may be widely separated. Failure to oppose and fix the fragments by operative means leads to nonunion. In fractures of long bones a part of the shaft

unite if properly reduced and immobilized for a sufficiently long period.

3 Excessive traction causes a gap wider than the osteoblasts can readily bridge

4. Poor circulation to the fractured fragments, especially if aseptic necrosis of a fragment occurs. This is most commonly seen in fractures of the scaphoid and neck



Fig 315—Pseudarthrosis of the humerus

may be missing (e.g., gunshot wounds). Such gap fractures fail to unite unless bone grafted or unless the ends are opposed.

2 Inadequate immobilization. Constant movement at the fracture line creates the conditions required for a false joint or pseudarthrosis. Practically all fractures will

of femur where the proximal fragment or femoral head is avascular.

5. Infection causes fibrosis and interference with the circulation and may also result in faulty immobilization

6. Absence of functional use of the part. Stress and strain both stimulate union if

achieved without shearing at the fracture line.

7. Old age, invalidism, poor nutritional state.

8. Pathological conditions of bone. When the fracture occurs through a malignant growth of primary or secondary nature.

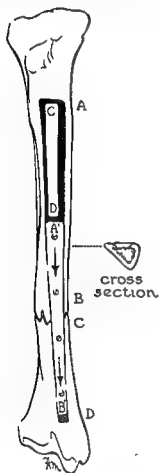


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Treatment.—When the presence of the calcified shadow is seen on x-rays after a joint injury, attention should be directed to prevent any passive stretching of the area. No operative procedures should be consid-

ered until the range of movement has become stationary and the x-rays show a dense shadow. If such constitutes a bony block, excision should be considered.



Fig 317—Myositis ossificans

Aseptic Necrosis

Following a fracture or dislocation, the blood supply to a fragment or area of bone may be less than the nutritional requirements. When such occurs the part undergoes aseptic necrosis. If in a position to be revascularized, the granulations grow into it and the bone is replaced by creeping substitution (Pheister).

Union of fractures where one fragment is avascular is greatly delayed. This is seen in cases of fractures of the scaphoid with a small proximal fragment; also in fractures of the femoral neck with death of the head.

Circulatory Complications

1. Acute bone atrophy—Sudeck.
2. Traumatic arterial spasm—Volkmann's Ischemic Contracture, Gangrene.
3. Fat Embolism.

Nerve Complications

Upper Limb.—

1. Brachial plexus—Fractured clavicle. Fracture dislocation of shoulder.
2. Axillary nerve—Dislocation of shoulder.
3. Radial nerve—Fracture of humerus.
4. Ulnar nerve—Fracture dislocation of elbow.
5. Median and ulnar nerves—Involved in Volkmann's contracture.
6. Median nerve—Irritated by anterior dislocation of the semilunar bone.
7. Digital nerves—In compound injuries of fingers.

Lower Limb.—

1. Sciatic nerve—In posterior dislocation of hip.
2. Peroneal nerve—In fractures of neck of fibula.

From pressure of plaster cast

REFERENCES

- Bick, Edgar M.: *History and Source Book of Orthopaedic Surgery*, New York, 1933, The Hospital for Joint Diseases.
- Bohler, Lorenz: *Treatment of Fractures*, ed. 4, Bristol, 1936, John Wright & Sons, Ltd.
- Campbell, Willis C.: *Operative Orthopedics*, ed. 2, St. Louis, 1949, The C. V. Mosby Company.
- Henry, Arnold K.: *Extensile Exposure Applied to Lumb Surgery*, Edinburgh, 1946, E & S Livingstone.
- Hilton, John: *Rest and Pain*, ed. 12, London, 1913, G. Bell & Sons.
- Jones, Reginald Watson: *Fractures and Joint Injuries*, ed. 3, Edinburgh, 1943, E & S. Livingstone.
- Keith, Arthur: *Menders of the Maimed*, London, 1919, Henry Frowde.
- Magnuson, Paul B.: *Fractures*, ed. 2, Philadelphia, 1936, J. B. Lippincott Company.
- Nicola, Toufick: *Atlas of Surgical Approaches to Bones and Joints*, New York, 1945, The Macmillan Company.
- Sharr, C. M., and Kreuz, Frank P.: *Manual of Fractures, Treatment by External Skeletal Fixation*, Philadelphia, 1943, W. B. Saunders Company.

CHAPTER XXXVI

JOINTS

H. F. MOSELEY, D.M.

The function of joints is to permit movement of the bones which make up the articulation. This movement occurs between the smooth articular cartilages that cover the bone ends. Secretion of lubricating fluid by the synovial membrane is controlled by functional requirements. The range of motion is determined by the shape of the articulating surfaces, the capsular and ligamentous attachments, and the musculature related to the bones concerned. Injury and disease have as their chief complication, interference with this gliding mechanism and consequent joint stiffness.

INJURIES

Penetrating Wounds

The superficial joints of the hand, foot, knee, or elbow may be contaminated by puncture wounds or opened by laceration. Foreign bodies such as parts of needles, pins, nails, glass, projectiles, clothes, wood and dirt may all form part of the injuring force.

Treatment.—The probability of infection indicates the serious nature of these lesions. At the earliest moment, the wound tract should be excised under appropriate anesthesia. Foreign bodies are removed and the joint irrigated with saline. Penicillin in aqueous solution is instilled into the joint cavity. The synovial membrane is closed without drainage, the superficial wound arranged for secondary suture on the fifth day, and a pressure bandage applied. The limb is splinted in a bivalved cast. Systemic penicillin is given until the possibility of infection has passed.

If the patient is seen later and the joint presents an effusion with local and general signs of infection, aspiration of the fluid and

daily instillation of 300,000 units of aqueous penicillin are performed. The bacteriologist determines the causative organisms and the chemotherapeutic or antibiotic agent required. For penicillin-sensitive organisms, penicillin can be injected locally into the joint together with intramuscular administration. Joints which in former years were destroyed by suppurative arthritis can thus be salvaged.

Ligaments

These are the capsular thickenings which prevent abnormal displacements of the bones. The ligament is blended with the periosteum and through this is attached to the bone by dense fibers penetrating along Volkmann's canals. Muscular balance prevents excessive tension on ligaments, and rupture does not occur unless these muscles are caught off balance or the force involved is greater than the protecting power of the muscles concerned.

The usual injury is a sprain which may be defined as a rupture of the fibers near the bony attachment. Sprains may be incomplete, involving only a few fibers, or complete, when all the fibers are ruptured. Sometimes the ligament avulses the piece of bone to which it is attached, giving a sprain fracture.

Treatment.—Incomplete sprains do not cause joint instability, and treatment is palliative. Adhesive strapping arranged to minimize tension on the affected fibers suffices. Cold applications for 8 to 12 hours, followed by heat, massage, or even Novocain infiltrations can be used to diminish pain.

Complete sprains and sprain fractures, as they permit joint instability, deserve as

serious consideration as fractures, and often produce more prolonged disability.

Complete sprains of the external lateral ligament of the ankle should be treated in a walking cast for 6 to 8 weeks. Sprains of the internal lateral ligament of the knee are usually incomplete, and strapping support suffices. When the ligament is completely torn in association with dislocation or valgus fracture dislocations, operative suture is probably best. Sprain fractures, with displacement of the medial malleolus, require open operative suture. Severe sprains of the wrist must always be differentiated from the fractured navicular by careful radiological studies.

Subluxations and Dislocations

When ligaments and capsule are sufficiently torn to permit joint instability, the articulating surfaces may displace partially or completely on each other. Partial displacements are usually self-reducing and are called subluxations. When complete separation of the surfaces occurs, the bones are locked in the displaced position by muscular spasm, and these complete persistent displacements are called dislocations.

If the initial treatment does not secure capsular and ligamentous healing, the instability persists and the joint is subject to recurrent subluxations or dislocations. The shoulder most frequently presents this clinical syndrome.

Occasionally dislocations are compound in nature or associated with a fracture. Damage to nerve or blood vessel may occur at the initial injury or result from forcible manipulative procedures. Pathological dislocations occur when tense effusion and inflammation destroy the ligaments and joint stability, thus permitting gravity and muscle imbalance to displace the component bones.

Treatment of Acute Dislocations.—Early reduction under anesthesia affording complete relaxation of muscular spasm is indi-

cated. The part should be protected until healing of the ligaments is secured. Re-education of joint movements and musculature is then essential.

Internal Derangements

Injury and disease may result in the presence of tissue which intrudes between the joint ends. Such tissue may be unorganized, as is found in chronic infective synovitis with formation of fibrin concretions called melon or rice seed bodies, or may be organized tissues of fibrocartilage, articular cartilage, or bone.

When completely detached, these fragments are called loose bodies and give rise to recurrent effusions, joint locking, and instability. By far the most common internal derangements occur in the knee from rupture of the semilunar cartilages.

Traumatic Synovitis and Hemarthrosis

Joints which are subjected to a direct blow or a severe sprain become painful and filled with an excess of thin synovial fluid. This condition is called traumatic synovitis and the collection of fluid a joint effusion. If the ligaments are torn or a fracture into the joint is present, hemorrhage may fill the joint giving an hemarthrosis. Unless severe tissue damage is present, the blood remains fluid. Both these conditions may follow joint operations.

Treatment consists in aspiration of tense effusions and application of pressure dressings. In the knee and ankle, protection from weight-bearing is advisable but joint and muscle exercises assist absorption and accelerate recovery.

Traumatic Arthritis

When injury to the bones or articular cartilage leaves a residual mechanical derangement of a joint or when capsular and ligamentous tears give permanent instability to the articulation, the wear and tear processes are accentuated and gradual destruc-

tion of the joint mechanism ensues. This destructive process is called *traumatic arthritis*.

Traumatic arthritis is most commonly seen in the weight-bearing joints, but especially occurs in the closely fitting joints such as the ankle and elbow. Aseptic necrosis of articular fragments such as the femoral head and proximal half of the scaphoid leads to this deterioration if protection from stress and strain is not secured during the revascularization of the ischemic fragments.

Treatment.—Traumatic arthritis is minimized by accurate reduction of fractures involving the articular surfaces together with the removal of loose fragments of bone or cartilage from the joint cavity. Avoidance of function by splinting is required in simple injuries of joints for a period of two to three weeks but in cases with aseptic necrosis the time factor will be governed by radiological evidence of revascularization.

INFECTIONS

Bacteria may be carried to joints by the blood stream, introduced directly by wounds, or extend from adjacent bone or soft tissue infections.

Acute Pyogenic Arthritis

The bacteria most commonly found are streptococci, staphylococci, pneumococci and occasionally *S. typhi*. These reach the synovial membrane by the blood stream and initiate an inflammatory reaction. A seropurulent exudate develops which gradually increases in quantity, distending the joint cavity. The limb takes up the position of comfort; i.e., that of maximum joint capacity; e.g., knee flexed 20°. The tension and inflammatory involvement of sensitive tissue result in great pain which is evoked by the slightest movement. Intensive muscle spasm is Nature's protective response.

Clinical Picture.—The patient presents the general reaction to infection with raised

temperature, pulse rate, and leukocytosis. The degree varies with the severity of infection.

Locally the joint is warm, swollen, and tender. All movements are resisted by the patient.

Diagnosis.—History of infection elsewhere, such as otitis media, tonsillitis, skin infections, or pneumonia suggests the site of origin and type of bacteria involved. Aspiration of the joint with microscopic examination and culture is essential.

Treatment.—Bed rest and general supportive measures are indicated. Aspiration of the joint fluid and replacement by penicillin should be repeated daily until resolution occurs. Intramuscular penicillin is also given. The limb is splinted in the position of greatest comfort.

When the infection has been overcome, gradual restoration of function can be obtained with physiotherapy.

Gonorrheal Arthritis

Infection of joints and synovial sheaths of tendons and bursae occasionally occurs and then usually in the third week after genital tract infection in the adult. Spread is by the blood stream. Such involvement is infrequent since the advent of penicillin.

The clinical picture differs from that caused by streptococci and staphylococci in the lesser degree of toxemia and systemic reaction. Locally the joint signs are similar. The process may involve several joints.

The arthritis is rapidly cleared by penicillin which has replaced previous therapies such as treatment by hyperpyrexia.

Chronic forms of Neisserian involvement of joints are associated with chronic synovitis giving recurrent effusions and with a form of articular rheumatism.

Tuberculous Arthritis

Arthritis caused by the tubercle bacillus is one of the most serious and disabling types of joint disease. Before the pasteurization

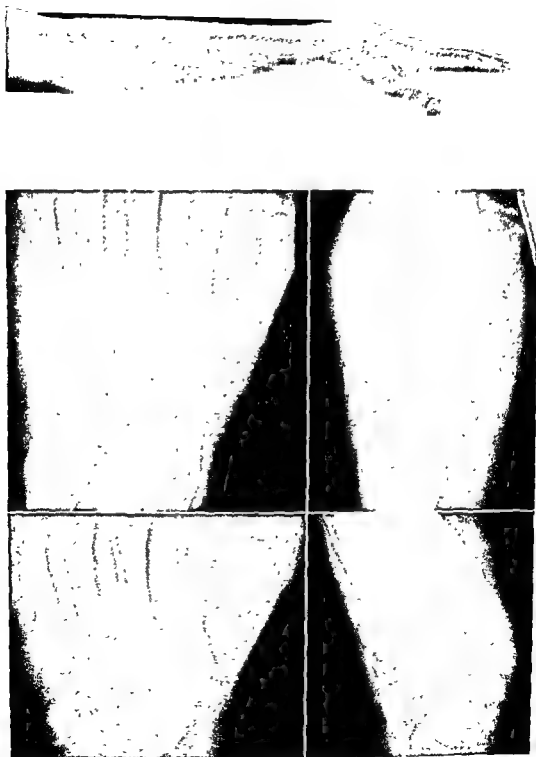


Fig. 318—*Tuberculous wrist*

Top, clinical appearance, center, x-ray appearance, bottom, after bone graft.

of milk and herd testing, the bovine bacillus was a frequent invader of joint tissues.

The bacilli reach the region by the blood stream from a primary focus in lymph gland or pulmonary tissue. The initial lesion may be in the juxta-articular bone with secondary involvement of the synovia or it may start in the lining membrane. An indolent, slowly progressing granulomatous thickening of the membrane occurs. The cartilage is eroded and sequestered by the carious process, and the ligaments are destroyed. Muscle spasm develops early and joint stiffness presents on clinical examination.

Aspiration of the joint with guinea pig inoculation is necessary for diagnostic purposes. In doubtful cases biopsy is indicated. X-ray examination in the fully developed case shows marked osteoporosis and loss of joint spacing.

Treatment.—

General.—The joint process is only part of the systemic disease and the general treatment of tuberculosis is essential. Long periods of observation in tuberculosis centers are necessary, with supportive therapy and training in the way of life required. Streptomycin has been found useful in joint tuber-



Fig. 319.—X-rays of tuberculous hip—early and late stages.

Diagnosis.—The patient may present the general picture of tuberculosis with loss of weight, afternoon elevation of temperature, and night sweats. If seen early, the joint signs, such as a limp when tired if a lower limb joint is involved, are minimal. X-rays at this stage may be negative. Tuberculin tests are positive. There is a relative lymphocytosis and raised sedimentation rate.

With progress of the disease, the joint becomes swollen and its movements are restricted. Local heat is not obvious. In the knee the thickened synovial membrane and enlarged joint contrasted with the wasted thigh and calf give the classical appearance to which the term "white swelling" is applied.

and is given in 1 Gm intramuscular injections daily for 60 to 90 days. When secondary sinuses are present, local application is utilized as well. Closure of sinuses can now be expected with this treatment.

Local.—The principle of treatment for joint tuberculosis is rest. In the young, conservative measures employing casts and braces are used. In the adult, resection of the joint and arthrodesis are indicated during a period when the bodily reaction to the disease is favorable as indicated by normal temperature, pulse, and gain in body weight.

Syphilitic Arthritis

The incidence of syphilitic disease of joints has been greatly diminished by active public



Fig 320—X-rays of Charcot's knee.



Fig 321—X-rays of Charcot's spine

health programs and free medical treatment. Cases, therefore, are rarely seen, but clinical types must be kept in mind for differential diagnosis.

The *early stage* is sometimes associated with arthralgia involving several joints, especially the knee, and may be accompanied by a chronic form of joint effusion. General antiluetic treatment affords relief.

The *late stage* presents the gummatous process in the tissues and this may cause an

Gummatous synovitis is also seen in children between 8 and 14 years who show the stigmas of congenital syphilis, such as interstitial keratitis, saddle nose, and Hutchinson's teeth. The process is usually bilateral and involves the knees in a painless effusion. The name Clutton's joints is given to this condition.

Painless nodules occur quite frequently around joints. These juxta-articular nodes vary from pea to walnut size.



Fig 322.—X-rays of degenerative arthritis of knee.

infiltration of the synovial membrane, giving an indolent form of *peri-arthritis*.

Diagnosis is made with recognition of the systemic disease. The Wassermann reaction is positive. The process must be differentiated from tuberculous synovitis. It will be noted that the syphilitic process tends to be bilateral and symmetrical and lacks the general systemic toxemia seen in tuberculosis. Biopsy of the synovial membrane may be required.

Treatment consists of general antiluetic measures. Relief from weight-bearing and splinting limits excessive function.

Charcot's Joints

Patients with *tabes dorsalis* and *syringomyelia* may present a rapid, painless destruction of one or more joints. In both of these diseases the *spinothalamic tracts* are destroyed, with loss of pain sensation. This is regarded as the causative factor in the



Fig 323 —X-rays of degenerative arthritis of knee with loose bodies, patella and loose bodies removed from knee

joint changes since injuries are not associated with pain, and protective muscular action is absent. Syphilitic arteritis in the vessels supplying the joint probably also predisposes to the degenerative changes.

The joints most frequently involved are the knee, hip, ankle, shoulder, and spine.

The clinical picture is made on the above findings in addition to the presence of the neurological signs of tabes or syringomyelia.

VARIOUS TYPES OF ARTHRITIS

Degenerative or Osteoarthritis

This is the most common form of joint disease and is the result of the wear and tear processes of daily life. The inherited constitution and vitality of the joint tissues are determining factors in its occurrence since the process runs in families. Injury, excessive weight and chronic postural strain, all play a part. It is characteristically seen in the patient over 50 years of age and most commonly involves the weight-bearing joints, i.e., knee, hip, and spine.

Pathology.—The soft tissues of the joint such as the ligaments and synovial membranes show the most marked changes. The ligamentous attachments are frayed and the synovial membrane thickened and hypertrophied into villous fringes. The cartilage over the weight-bearing or pressure points becomes fibrillated and gradually sequesters into the joint giving rise to loose bodies and recurrent effusion. Gradual deterioration of the whole joint then ensues.

Diagnosis.—Clinically the joint presents the signs of an irritative process. The movements are restricted in all directions and pain is elicited on manipulation. The knee takes up a position of flexion while the hip becomes flexed and adducted with some external rotation. X-rays show typical *lip-ping* and possibly loss of joint spacing. Examination of the hands may show the characteristic Heberden's nodes.

Treatment.—In the early stages, protection from excessive function, with diet to cause loss of weight in the obese, will help. Salicylates and physiotherapy relieve. When loose bodies form, arthrotomy with removal of debris sometimes gives excellent results. In late stages, orthopedic procedures such as arthroplasty or arthrodesis may be required.

Gouty Arthritis

Uric acid deposition in patients with gout may occur in any articulation. The great toe joint is the site of election. The knee, hip, lumbar spine, elbow (olecranon bursitis) all may present acute or chronic involvement.

Treatment.—Colchicine 1/120 gr. hourly till diarrhea occurs is most effective in the acute attack. Cold applications and protection from pressure or tension are fundamental in the local therapy. Dietary measures and salicylates are used between attacks.

Rheumatoid Arthritis

These cases are seen on the surgical wards only in the late stages, presenting the deformities of chronic polyarticular disease.

The characteristic fusiform swelling of the proximal phalangeal joints, the flexed swollen knees, flexed and adducted hips, and plantar flexed feet from ankle involvement are usually present.

Treatment of a surgical nature is directed to orthopedic procedures such as correction of deformity by manipulative measures and establishment of the capacity for ambulation by reconstructive and arthrodesing operation on the weight-bearing joints involved. The use of cortisone and ACTH may prevent or relieve these serious deformities.

REFERENCES

- Bennett, G. A., Waine, H., and Bauer, Walter: *Changes in the Knee Joint at Varying Ages*, New York, 1942, Commonwealth Fund.
Comroe, Bernard L.: *Arthritis and Allied Conditions*, ed. 4, Philadelphia, 1949, Lea & Febiger.

CHAPTER XXXVII

BURSAE

H. F. MOSELEY, D.M.

The word *bursa* is derived from the Latin *bursarius*, and signifies a sac. Such bursae or sacs are formed of a fibrous outer membrane and are lined by a layer of mesothelial cells which secretes the lubricating fluid corresponding to the synovial fluid in joints.

Bursae are found in constant sites in the human body and function to diminish friction and obviate pressure where one plane of soft tissue, such as skin or tendon, glides over another plane which usually presents a prominence of bone or tendon.

The *constant bursae* are those shown in the illustrations, but bursae may develop as the result of deformity producing a prominence over which the soft tissues must move or when an occupation subjects a certain area to repeated pressure and movement of soft tissue over the underlying skeletal parts. Such occasional sacs are called *adventitious bursae*. Some bursae communicate with joints such as the semimembranosus bursa behind the knee. This has been described as a posterior herniation of the synovial membrane. Other bursae may communicate with joints as the result of attritional changes in the intervening tendon or capsule. The best examples of such lesions are found in the subacromial, iliotrochanteric and prepatellar bursae.

Chronic bursal disease often necessitates surgical removal. This is followed by reformation from the residual bursal and adjacent areolar tissues in response to functional activity of the parts.

TRAUMATIC BURSITIS

Trauma affects the bursal mechanism in the same variety of ways and with the reactions corresponding to those described for joints.

The superficial bursae such as the olecranon and prepatellar bursae may be injured by a *penetrating* wound. Such open or compound injuries will be diagnosed by the presence of the wound which on examination leads to the lumen of the bursa and shows the discharge of mucinous fluid.

Treatment includes the careful excision of the wound edges and primary suture if seen within the period before infection has supervened on the contamination. The part should be locally splinted to secure rest and penicillin given to prevent the development of infection.

If the patient is seen after the infective process has developed in the wound, the bursal wall will be thickened and a seropurulent discharge will be present.

Treatment at first should be conservative with local rest by splinting of the part. The discharge should be studied as to its bacterial content and antibiotic sensitivity. Penicillin is best given immediately and changed to the appropriate antibiotic if the organism is not penicillin-sensitive. Some cases will require excision of the chronically infected bursa when the active infection has been controlled.

Trauma may also set up an acute traumatic bursitis as a result of a single contusion or from repeated unusual pressure and friction. A direct fall on the point of the elbow or knee may initiate the process in the olecranon or prepatellar bursa. Working in the supine position under a car or aeroplane with pressure on the elbows, and scrubbing floors on the knees, are common causes.

The irritated bursa fills with serous fluid containing more or less blood depending on the severity of the injury. In the contusion

injuries, fissured fractures of the underlying bone or rupture of the tendon attachment or periosteum may be present.

Treatment consists of rest to the part with application of cooling lotions for the first 8 to 12 hours to diminish the traumatic inflammatory process, followed by heat and massage and gradual resumption of function to accelerate the absorption of the inflammatory products. If the bursa is tensely swollen, aspiration of the fluid followed by a pressure dressing to diminish reformation, accelerates the recovery and affords relief of pain.

bodies of a fibrinous or even calcified nature may form and pressure on the area may be extremely painful. The bursa involved is subject to recurrent attacks of pain and the muscles in the area may exhibit painful protective spasm.

The treatment of the chronically thickened bursa presenting recurrent attacks of pain and swelling is excision. Conservative measures such as rest and protection from injury are justifiable in the early stages. Aspiration of the fluid is usually followed by recurrence.

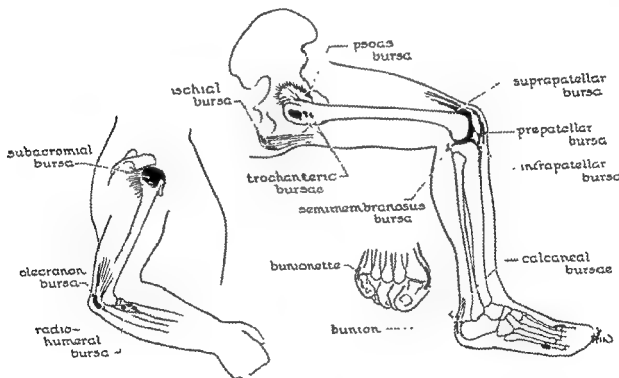


Fig 324—Bursae of the upper and lower limbs.

Although an acute trauma frequently produces bursitis, *chronic repeated trauma* of lesser degrees is the most common factor. The changes in the bursal wall are more gradual and in time a thickened fibrous wall with villous processes of the mesothelial lining develops. There is an excess of fluid present which is more serous in character than normal. The bursa tends to fill up and

INFECTIVE BURISITIS

Pyogenic infections of bursae may be primary and result from direct introduction by penetrating wounds or be metastatic due to hematogenous spread in cases of septicemia. Antibiotic treatment is usually adequate and drainage by aspiration is rarely required.

Gonococcal infection occurs secondarily to a genital tract infection and is controlled

Tuberculosis may develop in bursae usually by spread from adjacent bone or joint lesions. The trochanteric bursa is a typical site for such a tuberculous lesion and radiological examination will reveal changes in the underlying bone once the disease is well established.

Syphilitic bursitis is much less commonly seen today because of the early efficient treatment of the systemic disease. It is characteristic of the tertiary gummatous stage and the bursae most frequently involved are the prepatellar bursae. The process is typically bilateral and symmetrical.

The diagnosis is suggested by the presence of bilateral thickened bursae which are typically painless and have developed without adequate traumatic cause. The serological examination and clinical history will establish the presence of syphilis.

Treatment consists of the systemic treatment of syphilis.

METABOLIC BURSITIS

In this group of cases may be included, for descriptive purposes, bursitis due to deposits of uric acid and its derivatives and to deposits of the phosphate and carbonate of calcium.

Systemic gout with the recurrent precipitation of uric acid around joints leads to attacks of acute pain which are most frequent in relation to the great toe joint and related bursa. Sudden pain in relation to the olecranon bursa with diffuse swelling without apparent cause is sometimes due to gout.

Deposits of calcium phosphate and carbonate are most typically seen in the rotator cuff of the shoulder with secondary involvement of the subacromial (subdeltoid) bursa, although as in gout any joint or bursal mechanism may be involved.

In the cases of lesser severity of involvement by deposits of uric acid, calcium phosphate and carbonate, we have the explana-

tion for many syndromes loosely diagnosed as rheumatism, arthritis, neuralgia, and neuritis.

BURSAE OF THE UPPER LIMB

Three constant bursal mechanisms of the upper limb commonly present clinical syndromes. In order of frequency, these are subacromial (subdeltoid), olecranon, radiohumeral.

Subacromial (Subdeltoid) Bursitis

Involvement of this bursa is the most common cause of shoulder pain, and this is the "bursitis" so frequently discussed in the lay press.



Fig 325.—A calcified deposit which has ruptured into the subacromial bursa.

Knowledge of this entity was disseminated by Codman who demonstrated that the bursa is secondarily involved by inflammatory processes due to trauma, degeneration, and calcified deposits in the subjacent rotator cuff. The bursa functions as a secondary joint mechanism between the humeral head and the coracoacromial arch, and this gliding mechanism is necessary for the movements of rotation and elevation of the arm above the horizontal plane.

Contusions of the area from falls on the upper limb, degenerative changes, and calcified deposits in the underlying cuff produce irritation of the sensitive bursal lining. Depending on the severity of the inflammation are the intensity of pain and limitation of function.

In the typical case due to calcium deposition, the patient gives a history of recurrent rheumatic pain on change of weather, or overuse. The acute attack develops suddenly with inability to raise the arm, and sleeping on the affected side is impossible due to the severe pain. X-ray examination will indicate the presence or absence of calcium deposits.



Fig 326—Chronic olecranon bursitis

Treatment.—Conservative measures include support in a sling and rest to the part. Cooling applications or an ice bag afford more relief in the acute phase than heat. Analgesics are required for pain.

If x-rays show a large deposit of calcium, surgical excision during the acute phase is

best. Proper treatment for small deposits is a course of physiotherapy including heat and pulley exercises. X-ray therapy has many advocates.

In the chronic cases with and without the x-ray presence of calcium deposits, the use of diathermy, exercises, and the protection by wool garments in cold weather are advocated.

Olecranon Bursitis

Acute, recurrent, and chronic forms are seen and are related to varying occupations subjecting the elbow area to repeated friction, pressure or contusion. Miners, students, and mechanics are especially afflicted. The acute forms subside with rest but the recurrent and chronic types frequently necessitate excision of the bursa.

Radiohumeral Bursitis

This bursa is situated between the common extensor origin and the supinator brevis. Irritation of this mechanism by excessive pronation and supination is one cause of "tennis elbow." Rest, heat, and salicylates usually afford relief.

BURSAE OF THE LOWER LIMB

The constant bursae of the lower limb are more frequently subjected to trauma than those of the upper limb. The bursae commonly involved are: ischial, trochanteric, semimembranosus, metatarsophalangeal, psoas, prepatellar, calcaneal.

Ischial Bursitis (Weaver's Bottom)

A large bursa exists between the gluteus maximus and the ischial tuberosity and hamstrings origin. Constant friction and pressure from occupations involving the seated position for prolonged periods may initiate a bursitis. The chronically distended bursa may produce a swelling of considerable size. Treatment is excision.

Psoas Bursitis

A bursa exists in relation to the anterior capsule of the hip joint and communicates

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CHAPTER XXXVIII

FRACTURES AND OTHER DISORDERS OF THE UPPER EXTREMITY

H. F. MOSELEY, D.M.

CLAVICLE

The clavicle is so called from its resemblance to the clavicle of the musical score. It is highly developed in man, but may be congenitally absent, i.e., cleidocranial dysostosis. This bone is absent in certain running and jumping animals, e.g., the horse.

Functional Anatomy.—

The clavicle functions as:

1. A strut to keep the glenohumeral joint clear of the trunk, thus increasing the range of the hand as the effector organ of grasp.
2. A rigid attachment for powerful muscles.
3. The only bony connection of the upper limb and trunk.
4. A protection to the vital structures in the cervico- and thoraco-brachial outlets.
5. A cosmetic factor at the base of the neck.

Incidence.—Fractures of this bone are four times more common in the male than in the female. Sites of fracture: body—outer end—inner end.

Mechanism.—The usual cause is the transmission of force indirectly from a fall on the outstretched hand, elbow, or point of the shoulder. Fractures occasionally result from direct violence.

Fractures of the Body

The body is the area most frequently fractured. This portion marks the junction of the curvatures and has little protection from muscular or ligamentous attachments. Green-stick fractures are common in young children.

In complete fractures, the sternomastoid draws the inner fragment upward while the

weight of the limb causes the outer fragment to be displaced downward, forward, and medially.

Diagnosis is made on the history, the point of local tenderness or deformity, the localization of pain and crepitus to the clavicle on movements of the arm, and the positive x-ray findings.

Principles of Treatment.—

1. To brace the whole shoulder girdle so as to carry the outer fragment upward, outward and backward.
2. To depress the inner fragment.
3. To maintain reduction.
4. To continue active function of the hand, elbow and shoulder during immobilization of the fracture.
5. To check progress by x-ray.

Discussion of Present-Day Methods.—Unless some contraindication exists, most surgeons are content to use conservative methods and try to achieve good results by understanding the principles of reduction and using for support:

1. The three handkerchief or figure-of-eight bandage.
2. The figure-of-eight plaster cast
3. Adhesive strapping.
4. Bandage to shoulder cross.
5. Various commercial splints of light metal.

The three handkerchief method is the simplest to use, but it is necessary to explain to the parents of children or to the adult patient, the principles involved; the necessity to hold the shoulder braced upward and backward and the daily adjusting and padding of the bandages. Meticulous reduction and supervision are required in the young

with the joint in certain cases of degenerative and tuberculous arthritis. Chronic enlargement of this bursa is thus a lesion for differential diagnosis in swellings of the groin.

Trochanteric Bursitis

Several bursae exist in relation to the great trochanter of the femur. The large bursa deep to the tendon of the gluteus maximus may be acutely involved by calcified deposits in this area. Tuberculosis of the adjacent bone may cause a chronic process in this bursa.

Prepatellar Bursitis

This type was frequently seen in the domestics and cleaners of a previous generation. The acute form was presented by the uninitiated worker who attempted to scrub a large area from a fixed position. Today with modern apparatus, "housemaid's knee" is seldom seen. Bilateral disease of chronic nature with grossly thickened bursae should suggest syphilitic involvement. Serological examination if positive will confirm the diagnosis, and systemic treatment is curative.

The treatment of the chronically inflamed bursa is excision. The acutely inflamed bursa will subside with rest.

Semimembranosus Bursitis

This form of bursitis may be the cause of leg pain with stiffness in the movements of the knee. Examination of the popliteal space will reveal an oval tense swelling which disappears on flexing and becomes prominent on extension of the knee. It is typically seen in young boys of 10 to 15 years of age and if causing sufficient symptoms, it is best excised.

In older patients with arthritis of the knee, the bursa, if in communication with the joint cavity, will act as a blow valve and distend when the joint presents an effusion. Such distended bursae should not be excised. Treatment should be directed to the joint disease.

Calcaneal Bursitis

Bursitis may involve the bursa between the Achilles tendon and the bone or the subcutaneous bursa over the posterior prominence. Both types are due to ill-fitting shoes, and correction of this problem and protection from further injury suffice.

Metatarsophalangeal Bursitis

Inflammation of the bursa over the great (bunion) or small toe joint (bunionette) is a common complaint from ill-fitting shoes. Because of style and fashion female patients predominate. Most disorders are chronic. Treatment must be devoted to correction of the chronically deformed structures by orthopedic procedures. Properly fitted shoes must be prescribed.

REFERENCES

- Cherry, J. H., and Ghormley, R. K. Bursa and Ganglion, *Am. J. Surg.* 52: 319-330, 1941.
- Meyerding, H. W., and Van Demark, R. E. Posterior Hernia of the Knee (Baker's Cyst, Popliteal Cyst, Semimembranosus Bursitis, Medial Gastrocnemius Bursitis and Popliteal Bursitis), *J. A. M. A.* 122: 858-861, July 24, 1943.
- Monro, A. A Description of All the "Bursae Mucosae" of the Human Body, Edinburgh, 1788, Elliot.
- Moseley, H. F. Shoulder Lesions, ed 2, Springfield, Ill., 1952, Charles C Thomas.
- Wilson, P. D., Eyre-Brook, A. L., and Francis, J. D. A Clinical and Anatomical Study of the Semimembranosus Bursa in Relation to Popliteal Cyst, *J. Bone & Joint Surg.* 20. 963-984, 1938.

lady where the cosmetic result is important. Three to four weeks' immobilization is usually adequate.

insertion of the wire under direct vision will prevent penetration into important structures in this area.



Fig. 329—Fracture of the body of clavicle with typical displacement

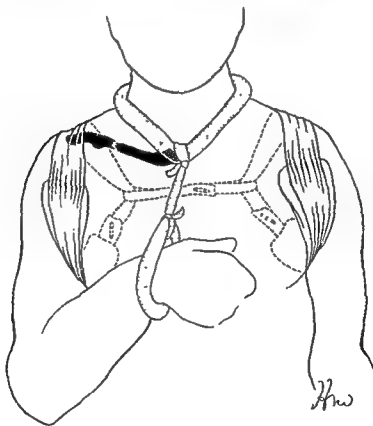


Fig. 330—Three handkerchief bandage applied for fractured clavicle.

Open operation is rarely required. If malunion must be prevented, the introduction of an intramedullary wire (Murray) is best. This must not be lightly undertaken. Exposure of the fracture site with

Fractures of the Outer End

Such fractures are protected from marked displacement by muscular and ligamentous attachments. Splinting is often unnecessary and may be minimal. When the fracture

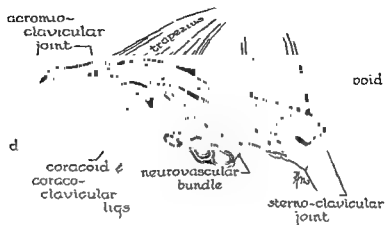


Fig 327 —Anatomy of the clavicle

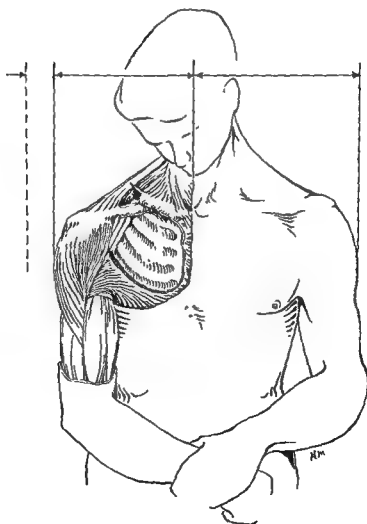


Fig. 328 —Fracture of clavicle showing typical deformity.

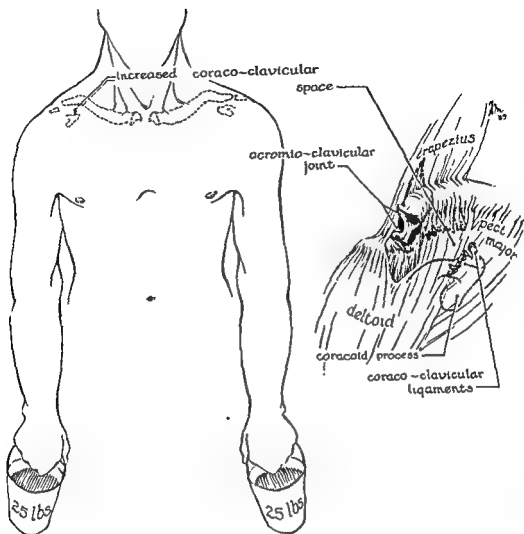


Fig 331.—Acromioclavicular dislocation.



Fig. 332.—Complete dislocation of acromioclavicular joint with increased coracoclavicular spacing indicating rupture of conoid and trapezoid ligaments

involves the acromioclavicular joint, resection of this portion eventually may be indicated

Fractures of the Inner End

Fractures of the inner end are protected from marked displacement by the muscular and ligamentous attachments. The routine methods of treating fractures of the body are therefore not usually necessary for fractures of the inner or outer end. A sling is sufficient.

Complications.—*Nonunion* is uncommon except after infection or unsuccessful operative intervention. Treatment consists of freshening the bone ends and employing internal fixation by onlay bone graft.

Stiffness of the shoulder is the most common complication in patients over 40. It is sometimes due to a concomitant injury to the shoulder joint, but more commonly results from immobilization of this joint during the therapy, and is best prevented by keeping the shoulder in use throughout.

THE CLAVICULAR ARTICULATIONS

Acromioclavicular Joint

The possibility of injuries of this joint is often overlooked.

Functional Anatomy.—The joint depends for its strength on the coracoclavicular ligaments as well as the capsular ligaments strengthened superiorly by the aponeurotic attachments of the deltoid and trapezius muscles. An intra-articular meniscus is present in 30 to 40% of cases. The clavicle may have an overriding articular facet with a loose joint arrangement or the clavicular facet may be vertical with a closely knit mechanism. The joint permits a small amount of movement in all directions, functioning in the range above 120° and especially in circumduction above 90° abduction.

Mechanism.—Minor sprains and subluxations may be produced by forces transmitted directly or indirectly. Complete dislocations occur when the patient falls on the point of

the shoulder. The whole shoulder girdle is forcibly depressed and the clavicle is arrested by the first rib. Continuation of the force causes rupture of the capsule and aponeurosis on the superior aspect followed successively by rupture of the trapezoid and conoid ligaments.

Diagnosis.—There is history of injury followed by pain over the superior aspect of the joint. In sprains and subluxations, forcible adduction of the arm across the chest will accentuate the pain which can be localized by palpation to the posterosuperior aspect of the joint. In doubtful cases, repeated circumduction of the arm above the horizontal plane, as in throwing a ball, will assist in localizing the pain and tenderness to this joint.

In dislocations the prominence of the outer end of the clavicle is obvious. X-ray examination confirms the displacement and is best done with the patient standing with a 25 pound weight in each hand. Rupture of the coracoclavicular ligaments can thus be readily diagnosed by increase in the coracoclavicular spacing.

Treatment.—In cases without rupture of the coracoclavicular ligaments, no special treatment is required. It is probable that, if symptoms persist for any length of time, the lesion is a derangement of the intra-articular meniscus.

In cases with complete dislocation, conservative treatment with the shoulder girdle elevated and downward pressure over the outer end of the clavicle for 6 weeks will result in good function in the majority of cases. This position can be secured and maintained by adhesive strapping and sling, or in a plaster spica. Some deformity usually persists.

In cases which are left with a painful joint after minor injuries, or with dislocation after conservative treatment and in certain acute dislocations, resection of the outer inch of the clavicle gives rapid and excellent results. This is recommended in preference to fascial or metallic repair.

The majority are impacted without serious displacement. When the fracture line passes to the inner side and the coraco-clavicular ligaments are ruptured, the outer fragment is depressed by the weight of the arm, and the clinical picture appears similar to that of anterior dislocation of the

In those with instability of the fragments or where other injuries necessitate bed rest, traction in the abducted position, together with elevation of the outer fragment by a band around the upper arm and arranged with gentle traction to the head of the bed for 3 to 4 weeks, is best.

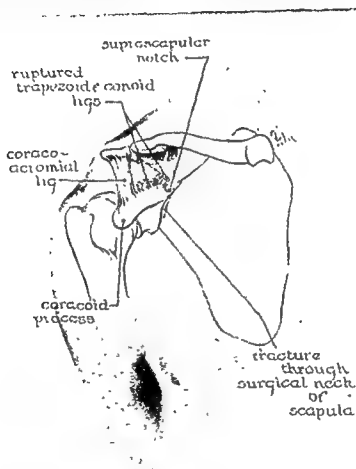


Fig 334.—Fracture of the surgical neck of the scapula

shoulder. The deformity readily disappears on pressing upward on the elbow, and reappears on release of pressure.

Treatment.—Cases impacted in fairly satisfactory position may be treated by early movement in the relaxed muscle position. When ambulating, the weight of the limb is taken by a sling arranged so as to elevate the arm. An axillary pad and encircling flannel bandage afford comfortable fixation.

Body

Fractures of this part are caused by direct violence or crushing injury which also frequently injures the underlying ribs. Because of the buffering and splinting action of the muscles arising from the three fossae, displacement is uncommon.

Treatment.—Aspiration of the hematoma, which often develops, may be indicated. Otherwise, treatment is by early function.

Sternoclavicular Joint

The sternoclavicular is an exceptionally strong joint and is the pivot for movements of the shoulder girdle, i.e., elevation, depression, protrusion, retraction, and circumduction. It is less frequently injured than the acromioclavicular joint. It constitutes the articulation between the upper limb and the trunk, and is involved in sprains, acute and recurrent subluxations and dislocations. Anteroinferior displacement usually occurs. Posterior dislocation has been described.

Mechanism.—Injury to this joint results from forcible pressure of the clavicle forward and inward. The capsule is torn and forward subluxation or dislocation occurs.

Diagnosis.—The prominence of the inner end of the clavicle is readily seen. Tomograms afford useful x-ray visualization of this joint.

Treatment.—In sprains and subluxations no special treatment is required. In complete dislocation, reduction is readily effected by bracing the shoulder backward and manually pressing the sternal end into place. This reduction is difficult to maintain and requires a plaster spica for 3 to 4 weeks with the arm at 90° abduction and 45° forward flexion with direct pressure over the joint in a backward and downward direction. Sometimes the figure-of-eight plaster bandage suffices as for a fractured clavicle.

In some acute and some recurrent dislocations, a fascial repair (Bankart) may be carried out or the sternal end of the clavicle may be resected, if the patient's symptoms of pain and functional derangement warrant operative treatment.

SCAPULA

Fractures of the scapula are not common but occur in the following parts:

1. Glenoid fossa.
2. Body.
3. Neck.
4. Acromion and spine.
5. Coracoid process.

Glenoid Fossa

Fragmentation of the bony rim may occur in anterior or posterior dislocations, especially if recurrent.

Fractures of the fossa are sustained when the humeral head is forced strongly against it by direct violence. Lesser injuries are contusions of the articular cartilage grading into multiple fissured fractures corresponding to the central dislocation found in the hip.

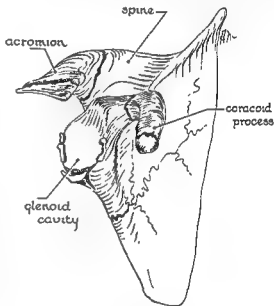


Fig. 333.—Composite diagram of type fractures of the scapula.

Treatment.—The treatment of fractures of the rim is part of that of the dislocation. Fractures of the fossa present a variable picture and are frequently associated with injury to the humeral head. In severe cases the problem is how to minimize the inevitable traumatic arthritis.

In the majority of cases, treatment by early movement is indicated. Where loose fragments of bone or cartilage exist in the joint or where associated ruptures of the rotator cuff are diagnosed, exploration of the joint should be carried out.

Neck

The fracture line may pass to the inner or outer side of the base of the coracoid.

neck compressed against the acromion as a fulcrum. Depending on the exact balance of forces, the distal fragment may be abducted or adducted in relation to the proximal fragment. Impaction is common.

Diagnosis is made on the history of a fall together with symptoms of pain and limitation of shoulder function. Palpation demonstrates tenderness at the surgical neck and radiological examination gives the final proof. Detailed views in anteroposterior and superoinferior planes are required. Stereoscopic views may be indicated.

Treatment.—In all cases impacted in satisfactory position and in most cases in

GREATER TUBEROSITY

Contusion Fractures

In cases of anterior dislocation of the shoulder, the greater tuberosity may suffer contusion from impingement on the anterior glenoid rim. This develops into the posterolateral notch seen in recurrent dislocations. The diagnosis is made by x-ray. Treatment is that for the dislocation.

Avulsion Fractures

These occur by contraction of the supraspinatus, infraspinatus and teres minor, often in conjunction with anterior disloca-



Fig 337—Impacted adduction fracture of surgical neck of humerus. A, anteroposterior; B, axillary view

patients over 45, the treatment advocated is that of immediate movement in the relaxed muscle position. A sling or collar and cuff is used for support.

In cases with marked displacement, the proximal fragment being abducted and externally rotated, in the younger patient (20 to 45), operative treatment with internal fixation by two nails gives excellent results. Early movement is encouraged postoperatively.

Treatment in abduction splints or in traction is required in some cases.

When the dislocation is reduced, the fragments usually fall into position as indicated by x-ray. If this does not occur, the arm may be splinted in abduction and external rotation, or the displaced tuberosity may be sutured in its normal position.

Avulsion of Supraspinatus Facet

Occasionally, only the supraspinatus facet is avulsed. This is diagnosed by x-ray and the patient's inability to abduct the arm. If it is displaced under the acromion, reduction is necessary to remove the bony block.

Acromion and Spine

Fractures of the acromion must be differentiated from epiphyseal separation in the young or from an ununited epiphysis by contralateral x-ray in later years.

The usual cause is direct violence, but sudden action of the condensed middle segment of the deltoid against resistance may produce the fracture. The superior periosteum and aponeurosis first tears allowing the fragment to hinge and, if the force carries on, this is followed by tearing of the inferior periosteum with separation of the fragment by deltoid traction. If the fragment is large, some deformity of the deltoid convexity is apparent.

Treatment.—These fractures usually heal by fibrous union. Where the fragment is depressed or separated, operative treatment with resuture is indicated to give a solid origin to the deltoid.

The same principles apply for fractures of the spine of the scapula.

Coracoid

Fractures of the tip are most commonly associated with anterior dislocations of the shoulder. Avulsion through the base may occur in place of rupture of the coracoclavicular ligaments in complete dislocation of the acromioclavicular joint.

Treatment in both types is part of that of the associated dislocation.

FRACTURES OF HUMERUS

Upper End

Fractures of the various portions of the upper end of the humerus occur as isolated injuries or may be associated with dislocations of the shoulder.

Surgical Neck

Incidence.—Fracture of the surgical neck is a common fracture, and occurs chiefly in the older age groups.

Mechanism.—It is caused by a fall on the abducted upper limb with the surgical

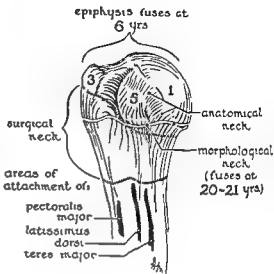


Fig. 335—Nomenclature of upper end of humerus

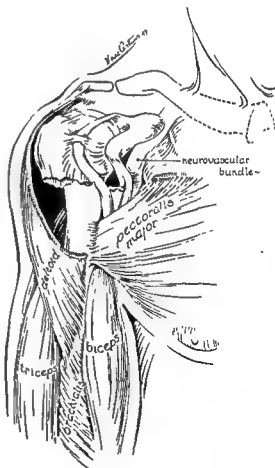


Fig. 336—Fracture of surgical neck.

DISLOCATIONS OF THE SHOULDER

The shoulder is the most common joint to suffer dislocation.

Functional Anatomy.—The shoulder joint is part of the shoulder girdle complex and does not function as an isolated unit. The glenohumeral joint is a ball and socket joint possessing great mobility. The middle segment of the deltoid and the supraspinatus act as a team abducting the humerus on the scapula with simultaneous descent of the head in the fossa. This is the last shoulder function evolved and is the first lost after injury or disease.

Classification.—

- | | |
|-------------------|----------------------|
| A. Simple | { Anterior |
| | { Posterior |
| B. Recurrent | { Anterior |
| | { Posterior |
| C. Complicated by | { Fracture |
| | { Rupture of Rotator |
| | { Cuff |
| | { Nerve Injury |

Mechanism of Injury.—Anteroinferior dislocation results from a fall on the upper limb in such a way that the arm is abducted and externally rotated levering the humerus against the acromion as a fulcrum out of the glenoid fossa. Sometimes dislocation occurs from a direct force from behind.

Posterior dislocation occurs with the arm in adduction and inward rotation with a force transmitted through the humerus in an upward and backward direction.

Post-Traumatic Anatomy.—Operative and autopsy findings tend to show that the usual lesions are:

1. Separation of the anteroinferior or posterior glenoid labrum in the respective anterior or posterior dislocations.
2. Separation of the capsule from the inferior aspect of the neck of the humerus.
3. Separation of the capsule and subscapularis tendon with or without the lesser tuberosity.

Diagnosis.—Anterior dislocations are diagnosed by the history of the accident, the location of pain, loss of function of the shoulder, and loss of the rounded contour of the deltoid. Palpation reveals the absence of the head under the acromion and its forward position. The axis of the humerus runs toward the middle of the clavicle.

Radiological examination confirms the clinical opinion and should be carried out routinely to ascertain whether an associated fracture is present.

When the shoulder has been dislocated longer than 3 to 4 weeks, the possibility of reduction by closed manipulation becomes doubtful and the condition may be diagnosed as a *chronic dislocation*.

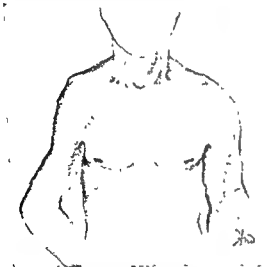


Fig. 340—Anterior dislocation of shoulder showing flattening of deltoid with axis of humerus directed toward middle of clavicle.

The head of the humerus, covered by the rotator cuff, revolves in relation to the coracoacromial arch with interposed subacromial bursa. This mechanism is of great clinical significance and merits the name superior humeral joint. As the humerus must externally rotate during abduction, lesions in this area restrict abduction.

In abduction and forward flexion, movements of the humerus, scapula, and clavicle occur simultaneously and rhythmically, i.e., scapulohumeral rhythm. Clinical examination should center on alterations in this rhythm rather than on localized restriction of movements of the arm.

Two forms of treatment are possible:

1. Resuture of the torn cuff with or without removal of the bony fragment.
2. Splintage in abduction for 4 weeks

Treatment.—After reduction of the dislocation the limb is rested in a sling for 3 to 4 weeks and this is followed by gradual re-education of shoulder function.



Fig. 338.—Displaced surgical neck fracture before and after internal fixation. Lateral views showed anterior displacement of humeral shaft which was corrected at operation. A Before operation. B After internal fixation using two Vitallium nails.

Separation of Upper Humeral Epiphysis

Incidence.—These occur in children between the ages of 5 and 15 years.

Diagnosis is made on a typical x-ray picture associated with the presence clinically of a severely injured shoulder.

Treatment.—In the displaced fracture, open operation is advocated with the use of Kirschner wire fixation for 3 to 4 weeks, followed by its removal.

When specialist facilities are lacking, fluoroscopic reduction and immobilization in adduction and internal rotation may be employed.

Lesser Tuberosity

Fractures of this tuberosity occur in rare cases of anterior dislocations of the shoulder and represent an avulsion fracture caused by the subscapularis.

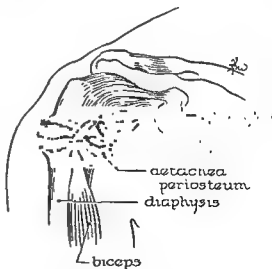


Fig. 339.—Diagram showing separation of upper humeral epiphysis.

Posterior dislocations are not suspected and are most frequently overlooked, especially in fat patients.

Diagnosis is made on the inability to abduct or externally rotate the arm which is

Manipulation under an anesthetic may be the final diagnostic point.

When the patient gives a history of several dislocations, the diagnosis, *recurrent dislocation*, is made.

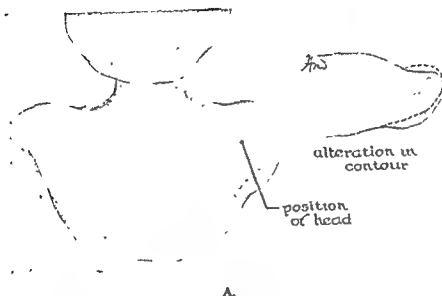


Fig. 343.—A Posterior dislocation of right shoulder showing alteration in contour.

B X-ray showing anterointernal notch and posterior dislocation of shoulder. (From "An Atlas of Shoulder Dislocations" by H. F. Moseley, Courtesy of Abbott Laboratories)

held in internal rotation. The head may be palpated in the subspinous position where maximum tenderness is present.

Routine x-rays are often inconclusive and stereoscopic views and superoinferior views are a necessity.

Treatment

Acute Anterior Dislocation

Reduction is best carried out under the relaxation provided by general anesthesia but can be effected without this assistance,

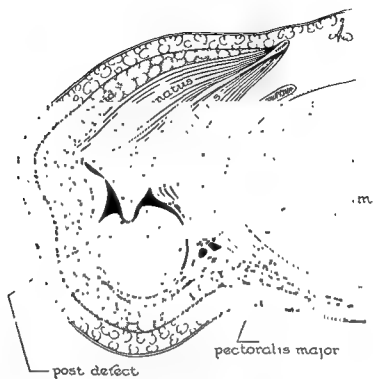


Fig 341—Cross section of anterior dislocation of shoulder



Fig 342—Anteroinferior dislocation of shoulder.

head. There is a tendency to instability of reduction and operative suture of the capsular defect may be indicated.

Recurrent Dislocation

Surgical treatment for this condition is becoming standardized. Repair of the capsule torn from the glenoid rim (Bankart) is performed. Silk sutures through drill holes, staples, fascia (Gallie), bone graft or metallic rim are different materials employed.

Complicated Dislocations

Dislocations may be complicated by fractures of the greater tuberosity, surgical or anatomical neck, lesser tuberosity or glenoid rim. Diagnosis is made by x-ray examination.

Treatment.—The principle of treatment is the reduction of the dislocation and fracture as well. In the adult patient open operation is frequently needed.

Ruptures of the rotator cuff should be suspected in patients over 50 who do not regain the power of abduction and where the axillary nerve is intact as indicated by deltoid contraction against resistance. Operative suture is required.

Dislocations of the shoulder may be accompanied by nerve injuries.

1. Axillary nerve.
2. Posterior cord.
- 3 Complete brachial plexus.

Sometimes nerve injury results from the excessive force used in reducing the dislocation. These nerve injuries usually recover on conservative therapy, as the injury is one of contusion or stretching.

Rupture of the axillary vessels has been noted, especially in manipulation of the chronic dislocation. Loss of limb and death have resulted.

LESIONS OF THE SOFT TISSUES

Ruptures of the Rotator Cuff

The term rotator cuff is applied to the musculotendinous envelope formed by the

supraspinatus, infraspinatus, and teres minor attached to the greater tuberosity and the subscapularis attached to the lesser tuberosity. The former are external rotators, the last an internal rotator of the humerus. The tendons of these muscles fuse with the capsule before insertion on the humerus. They function as a fine adjustment in the shoulder mechanism.

Incidence.—Ruptures of the rotator cuff are frequent injuries in patients over 50 but usually fail to receive adequate medical attention.

Mechanism.—These injuries result from falls and often accompany dislocation of the shoulder. It is rare to find complete ruptures before the 5th decade and the predisposing factors are the degenerative changes of ageing.

Clinical Picture.—The patient is usually a laborer who, on the occasion of a fall or severe strain, feels something snap in the shoulder. This is followed by acute pain which increases during the following 12 to 24 hours. He is unable to abduct the shoulder beyond 60 to 70°, and this is accomplished by the shrugging mechanism. Examination in the acute phase reveals tenderness over the tendon insertion. The arm can be lifted passively to 90° but may fall to the side ("drop arm sign") or show marked weakness in maintaining abduction against resistance. Injection of 10 c.c. 2% Procaine into the area of rupture relieves the pain, but the weakness in attaining or maintaining abduction persists.

Clinical Course.—Untreated, the pain diminishes and in time the patient regains the use of the arm. If the patient persists in retraining, full movement in the sagittal plane and up to 120° in the coronal plane may be attained. The weakness of the shoulder arm mechanism persists and the joint is subject to recurrent bouts of pain on overuse.

Post-Traumatic Anatomy.—Ruptures which involve the full thickness of tendon

It should be done at the earliest possible moment.

The principle of reduction is to make the head retrace its passage with the least possible further injury to the soft tissues.

Three methods are available:

1. Gentle traction in abduction to 140° and external rotation, i.e., the position of dislocation, associated with direct backward pressure on the head as the arm is adducted and internally rotated.

An alternative is to permit function of the joint but prevent for 4 weeks abduction above 90° or external rotation. The power of the intrinsic muscles is maintained by resistance exercises in this range.

Chronic Dislocation

Reduction has been accomplished up to six weeks following injury, but after 3 to 4 weeks may be dangerous because of adherence of the axillary artery and brachial



Fig. 344.—X-ray of fracture-dislocation.

2. Hippocratic Method: Traction on the arm with the unbooted heel in the axilla as a fulcrum

- 3 Kocher's Method: Long axis traction with the arm first externally rotated, then adducted across chest and finally internally rotated.

Postoperative Therapy.—The most certain way to prevent recurrence is by bandaging the arm to the side for 4 weeks and following this by re-education of the limb musculature and movements.

plexus to the productive scar tissue in front of the joint. If the patient is young enough to justify operative treatment, this would be carried out through the anterior approach to the joint. Should the dislocation be of long standing and function adequate, operation should be avoided

Posterior Dislocation

Reduction can be effected by traction and external rotation in adduction associated with outward and forward pressure on the

Treatment.—Unless contraindicated by age or general health, the ideal treatment is operative suture at the earliest moment.

the limb can be in a sling without tension on the suture line. Early movements in the relaxed muscle position can be carried out



Fig 346 —The shrugging mechanism shown on left shoulder. Note that by bending the trunk to the right, the elevation of the arm is increased.

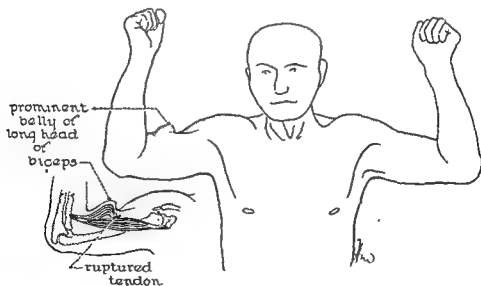


Fig 347 —Rupture of long tendon of right biceps in a case of degenerative periartthritis

The tendon is sutured to a trough cut near its original insertion. Postoperative care consists of rest in an abduction splint or in a sling. It is best to repair in such a way that

but active abduction, lifting the weight of the arm, should not be permitted for at least 4 to 6 weeks. The whole period of re-education requires 3 to 6 months, but the bene-

and capsule are called *complete* and those involving some fibers on the joint or bursal surface or in the substance of the tendon are called *partial ruptures*. Complete ruptures usually involve the supraspinatus tendon from the bicipital groove backward. Often the three external rotators are ruptured or the subscapularis may be involved as well.

floor of this the long tendon of the biceps is found. This tendon gradually wears by attrition against the acromion and coracoacromial ligament. This is the commonest cause for rupture of the biceps tendon.

Diagnosis.—Diagnosis is made on the history of a severe injury in a patient over 50 years of age who immediately has inability

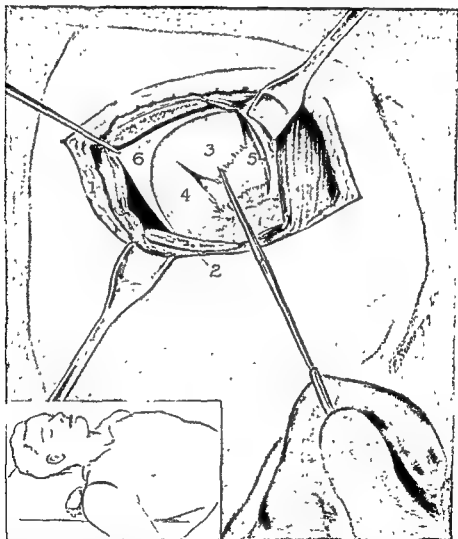


Fig. 345 —Rupture of the rotator cuff.

1 Skin and subcutaneous tissue 2. Sectioned acromion 3. Supraspinatus tendon
4. Infraspinatus tendon. 5. Long tendon of biceps 6. Roof of subacromial bursa

Such are called *massive* ruptures. The rupture occurs through a critical zone just proximal to the bony insertion thus leaving a stub of tendon on the bone. Untreated, the edges become smoothed off and the tendons retract leaving a triangular defect. In the

to abduct the arm X-rays are negative for fracture or dislocation. The axillary nerve is not paralyzed. Procaine injection demonstrates marked weakness in attaining or maintaining abduction. The drop arm sign indicates massive rupture.

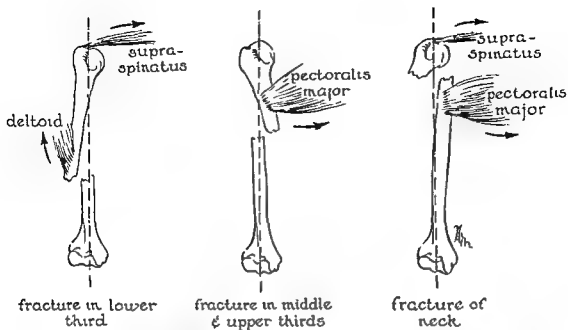


Fig. 348—Influence of muscular pull on fractures of the humerus at different levels.



A.

B.

Fig. 349.—A X-ray of fractured humerus treated in hanging cast. B Clinical union with improved alignment.

fits of operation are most clearly seen after 1 year. When patients are seen at a late period with weak and painful shoulders and with restricted movement, a trial of the benefit to be obtained by re-education of the shoulder with pulley exercises should be carried out before reconstructive surgery on the torn tendons is considered.

Rupture of the Long Tendon of the Biceps

Rupture of the tendon of the biceps may occur in the intra-articular portion, at the junction with the muscle belly or at its insertion. The first is by far the commonest type.

Incidence.—As for ruptures of the rotator cuff, the biceps tendon is ruptured in the laboring class and in the age group over 50 years.

Mechanism.—The rupture may occur spontaneously from years of gradual attrition involving also the supraspinatus tendon. In other cases it occurs simultaneously with rupture of the supraspinatus tendon when a heavy weight is suddenly lifted above the level of the shoulder.

Clinical Picture.—The patient feels a sudden pain in the shoulder with a snap and notes a lump in the arm. The pain may be minimal in the attrition rupture in senile cases.

Examination reveals tenderness in the bicipital groove and restriction of shoulder movements. Crepitus is usually present on rotation of the humeral head. Signs of rupture of the rotator cuff are noted.

On supination of the forearm against resistance, the belly of the long head will harden, showing loss of its elongated shape. When relaxed, the loss of the tension of the tendon permits increased mobility to the examining fingers.

Diagnosis.—Diagnosis is readily made on the history of injury and presence of the swelling in the arm. The possibility of concomitant cuff rupture must be considered.

Treatment.—In old patients, reparative treatment is elective. In the active laborer,

suture of the tendon in the bicipital groove with plastic repair of the cuff is indicated. The patient should not be permitted heavy use of the limb for 8 to 12 weeks.

Recurrent Dislocation of the Biceps Tendon

This is a rare condition in which the tendon slips forward over the lesser tuberosity and occurs where repeated stretching or one severe injury has weakened the bicipital sheath at the top of the groove.

Diagnosis is made by feeling the tendon slipping forward as the humerus is externally rotated.

Treatment is directed to transection of the tendon and fixation of the distal end in its groove.

SHAFT OF HUMERUS

The shaft consists of that portion between the surgical neck and the condyles.

Mechanism.—These fractures may be caused by direct or indirect violence. The resultant injury is a transverse, spiral or comminuted fracture.

The deforming forces are the fracturing violence and muscle pull.

Fractures occurring above the deltoid insertion have the upper fragment drawn inward by the pectoralis major and latissimus dorsi, while the lower fragment is displaced outward by the deltoid. Fractures below this level have the upper fragment drawn outward by the deltoid in relation to the lower fragment. The sling position most commonly used for splintage places the lower fragment in internal rotation and adduction.

Diagnosis.—The arm is useless in the complete fractures, with local tenderness, swelling, and deformity marking the site. Radiological examination gives the exact details of the bony injury and should include three dimensional studies.

Examination should include a study of the muscles supplied by the radial nerve. *Wrist drop* is the characteristic finding when this nerve is injured.

Mechanism.—The force is usually indirect through the hand, forearm to elbow with posterior displacement of the lower fragment of the humerus. The elbow is almost completely extended.

The reverse fracture may occur by a direct blow from behind on the flexed joint.

Post-Traumatic Anatomy.—The line of fracture runs upward and backward. The sharp lower end of the humeral shaft may injure the brachial vessels or median nerve.

by x-ray. Immobilization is maintained for three weeks. Re-education of function follows.

Condylar Fractures

Fracture of the medial or lateral condyle or both occurs in the adult as a result of direct or indirect violence. The fracture most frequently involves the lateral condyle.

Cases with minimal displacement require rest for three weeks. In the displaced frac-



Fig 352 —X-ray of supracondylar fracture with forward displacement of the lower fragment. The more common displacement is posterior.

Clinical Picture.—The patient holds his elbow at 140°. The differential diagnosis must exclude a posterior dislocation. Gross swelling may obscure the bony landmarks of the epicondyles and olecranon which are in their normal relations to each other in contradistinction to the findings in posterior dislocation. X-ray examination is conclusive.

Treatment.—Reduction is effected at the earliest moment under general anesthesia. Gentle traction with direct pressure downward and forward on the olecranon is used. Splintage is arranged in the maximum of flexion permitted by the circulatory condition of the limb. Reduction is confirmed



Fig 353 —Intercondylar T fracture of humerus.

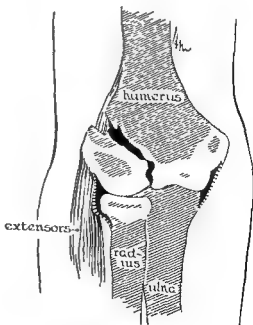


Fig. 354.—Lateral condylar fracture.

Treatment.—Conservative methods usually suffice. In spiral fractures and in the transverse fractures after manipulative reduction, a spica or sugar tong cast is applied. In fractures in the lower half, a hanging cast can be used.

When reduction has been unsuccessful, traction is employed.

in the spiral type of fracture and in the young patient.

Complications.—*Nonunion* may occur in the transverse fractures in the upper and lower thirds as a result of inadequate treatment. *Injury to the radial nerve* is the most common complication. *Stiffness of shoulder and elbow with vasomotor disturbance* of the

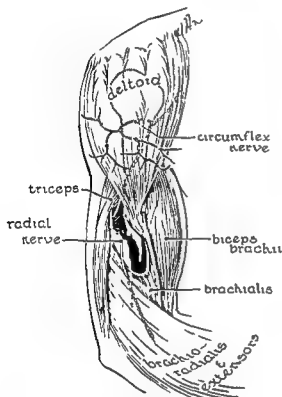


Fig. 350

Fig 350—Fractured humerus with injury to radial nerve.

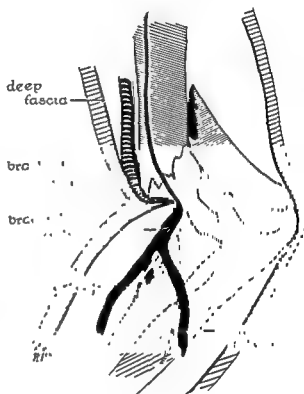


Fig. 351.

Fig 351.—Diagram of supracondylar fracture with mechanism of Volkmann's contracture (After Bunnell)

Traction methods are advised in small hospitals in preference to open operation with internal fixation. The latter is the method of choice in the hands of specialists when manipulative reduction is unsatisfactory and when injury to the radial nerve is present. The best approach is that of Henry extending along the outer aspect of the biceps.

The time for healing in fractures of the humerus is about 6 to 12 weeks, being less

in the spiral type of fracture and in the young patient. These are best prevented by functional use of the limb and avoidance of passive splinting.

FRACTURES OF THE LOWER END OF THE HUMERUS

Supracondylar Fracture

This is the most common fracture of the lower end of the humerus. It occurs most frequently from 10 to 15 years

Fissured fractures with displacement and comminuted fractures are best treated by resection of the head and removal of all loose fragments at the earliest moment. Postoperative therapy consists of gentle movements, but traumatic arthritis is minimized by avoiding heavy work for 6 to 12 weeks.

Post-Traumatic Anatomy.—This injury should be compared with that of the patella. Both involve the extensor mechanism of a hinge joint. The indirect type of fracture is transverse, and when separation is present the aponeurotic attachment of the triceps on each side is torn. Direct violence results in a comminuted type of fracture.

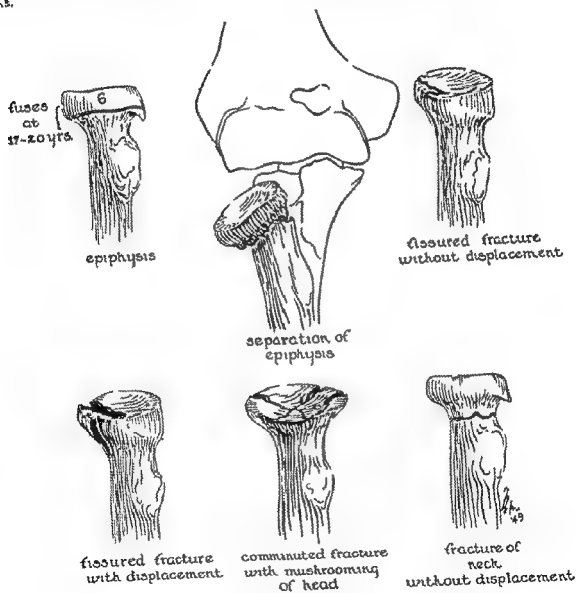


Fig. 355.—Type of fractures of head and neck of radius.

FRACTURES OF THE OLECRANON

Mechanism.—Fractures of the olecranon may be caused by indirect violence through the triceps or by a direct blow to this prominence.

Diagnosis.—Clinically the area is swollen and tender. The gap between the fragments may be seen or palpated.

Treatment.—Stable transverse or comminuted fractures without displacement may

tures, accurate anatomical reduction is indicated at the earliest moment and this is best carried out by open operation and internal fixation. In all cases plaster of Paris affords the most useful splinting

Kocher's Fracture

This rare but important fracture consists of the separation of a semilunar fragment of articular cartilage and bone from the capitellum. This may be associated with other fractures of the elbow such as that of the radial head. The fragment is usually displaced into the front of the joint.

Diagnosis is by x-ray and the treatment is by excision of the fragment

Fractures of the Epicondyles

Fractures of the epicondyles occur in the young, as epiphyseal separations, or in the adult, from direct violence or by avulsion. The medial epicondyle is the more frequently separated. Conservative therapy suffices except in gross displacement of the medial epicondyle with injury of the ulnar nerve when operative intervention is indicated. This consists of anterior transposition of the nerve with removal or internal fixation of the fragment. When the epicondyle is retained in the joint after reduction of a dislocation, faradic stimulation of the flexors usually secures reduction

Separation of Lower Humeral Epiphysis

The whole lower epiphysis may be separated at the age of 12 or the capitellar epiphysis at an earlier age.

Diagnosis is made on clinical and x-ray examination. Replacement by manipulative means may be successful. If not, open operation is indicated to prevent growth disturbances. There is a tendency to premature synostosis of the separated lateral condylar epiphysis and cubitus valgus results. Ulnar neuritis is a late complication.

HEAD AND NECK OF RADIUS

Fractures of the head of the radius are disabling because of the subsequent limitation of pronation and supination of the forearm and hand. They are often associated with dislocation of the elbow.

Mechanism.—Fractures result from indirect violence through the hand transmitted up the radius to the head which is impacted on the capitellar surface of the humerus. Direct violence may also cause fractures to the head alone or to all bones forming the elbow joint.

Post-Traumatic Anatomy.—Radiological examination indicates the fissuring of the bone, but the cartilaginous damage of the radius and capitellum must be surmised. The types of fracture are indicated in the diagram.

Diagnosis is made by noting the swelling over the head of radius together with local tenderness and pain accentuated by rotation. X-rays are conclusive and indicate the extent of the injuries.

Treatment.—

Separation of the Upper Radial Epiphysis—If displacement is minimal, no special therapy is required. If displaced, open operative reduction with immobilization in full flexion for 10 to 14 days is indicated. The epiphysis must never be removed before the age of 15 because of the inevitable growth disturbances.

Fracture of the Neck of the Radius.—In the presence of marked displacement, operation is indicated. If the head can be anatomically replaced and sutured, this should be done. Usually the whole head and the loose fragments must be removed, and the neck covered with soft tissue to give a smooth surface.

Fractures of the Head.—Fissured fractures without displacement require immobilization in flexion for 3 weeks to give healing to the fractured joint surface and fixation of loose fragments of cartilage.

It is characterized by tenderness over the lateral epicondyle and pain radiating over the extensor aspect of the forearm. There is weakness and pain on extending the wrist against resistance and any use of the limb accentuates the discomfort.

The lesion is a sprain of the common extensor origin and may be associated with deposition of calcium. Rest together with heat and salicylates is effective usually. If the condition becomes chronic, subperiosteal resection of the common extensor origin is the best therapy. Calcified material should be removed.

Stiff Elbow and Traumatic Arthritis

This is an all too frequent complication of elbow injuries. The elbow is a closely fitting joint and does not react favorably to intra-articular trauma. All operative procedures should remove any mechanical irregularity or loose fragment. This joint does not permit passive movements of a forcible character which usually result in increasing stiffness. It should, if possible, be splinted in flexion above the right angle and in re-education the progress should be accurately charted after measurement of range by the goniometer. Unless the range is progressively increasing without loss of flexion, the therapy is either being instituted too early or is excessive. There may be an intra-articular derangement causing delay in progress and the condition should be reviewed.

DISLOCATIONS

Dislocations occur in this joint more commonly in the young and as a result of indirect forces transmitted through the forearm and hand. The majority (80%) are posterior dislocations and may be complicated by fractures of the medial epicondyle, head of radius, or coronoid process.

Posterior Dislocation

Mechanism.—Posterior dislocation is caused by a fall on the outstretched hand, the force being transmitted upward with the

elbow dislocating by sudden hyperextension and contraction of the triceps.

Post-Traumatic Anatomy.—The displacement of the ulna is upward and backward. The coronoid process may be fractured. The capsule is torn anteriorly and on each side from the humerus.



Fig 357.—Photograph of patient with posterior dislocation.

Clinical Picture.—The patient holds the painful and swollen elbow at 140° to 160° . Flexion is impossible. The relation of the epicondyles and olecranon is altered and the olecranon process is very prominent. X-ray examination indicates the displacement and associated fractures.

Reduction is best effected under general anesthesia. Gentle traction on the hand with the elbow at 160° , with direct pressure on the displaced olecranon in a downward and forward direction, usually reduces the displacement with ease. The limb should

be treated by early function associated with local hot moist applications to diminish the swelling.

Fractures with separation require exploration and suture. Suture should include the aponeurotic tear as well as the osseous fragments. In the older patient excision of the fragments is often the method of choice.

Immobilization may be in a sling or light plaster cast at 90°. Movements may be encouraged at an early stage, 7 to 10 days, and excellent results are obtained in 6 to 12 weeks.



Fig 356.—Separated fracture of olecranon.

Complicated Injuries of the Elbow

All varieties of injury are found in this region. One of the most difficult is that in which the projecting elbow is struck by a passing vehicle—so-called “side-swipe” fracture. This may be compound and usually consists of a comminuted fracture of all three bones.

LESIONS OF THE SOFT TISSUES

Myositis Ossificans

This is one of the most common complications of injuries of the elbow. The an-

terior capsule, brachialis anticus insertion and annular ligament are most frequently involved. (See General Section on Fractures.) The condition is aggravated by passive movements, especially by forcible extension in the early stages after injury.

Treatment.—Function may be encouraged while the range of motion is increasing. When suspected, the course of the ossification should be followed by repeated x-rays, and gentle active movements only must be permitted. If the case is seen late with a dense area of calcified tissue limiting motion, operative removal must be considered.

Volkmann's Ischemic Contracture

This occurs most frequently after elbow injuries and is due to an interference to the circulation of the forearm muscles. The cause may be due to a spasm of the brachial artery or an obstruction to the venous return.

Treatment.—Prevention by early reduction of dislocations and displaced fractures and by elevation of the limb to encourage venous return is best. When the onset is suspected, because of pain in the forearm, swelling of fingers and absent radial pulse, hot moist packs and sympathetic block may help. If this fails, exposure of the vessels is required. After the chronic contracture is established, even extensive reconstructive surgery leaves considerable impairment of function.

Secondary Ulnar Palsy

This follows fractures of the lateral condyle in the young with premature synostosis and development of cubitus valgus. The ulnar nerve is gradually involved in a traumatic neuritis which develops years after the injury. Clawing of the 4th and 5th fingers with typical sensory changes is present.

Treatment consists of anterior transposition of the ulnar nerve.

Tennis Elbow

Tennis elbow is a clinical syndrome with several underlying causes.

be splinted in as much flexion as the circulatory state will permit. Dislocations of more than 2 weeks' standing usually require open operative reduction.

Postoperative Therapy.—Gentle active movements can be carried out almost immediately as the reduction is stable. Movements should consist of active flexion. If the range of motion progressively increases, function may be augmented. A full range may be secured in 4 to 6 weeks. Alternatively, plaster immobilization in the flexed position for 3 to 4 weeks may be employed.

Posterolateral Dislocations

These differ from the above in the lateral displacement and x-rays may show avulsion of the medial epicondyle. After reduction, the medial epicondyle sometimes remains within the joint. Operation with suture or removal of this fragment and with anterior transposition of the ulnar nerve is then indicated. The ulnar nerve may be injured in this dislocation. These cases are best treated in plaster casts as the reduction may be unstable.

Monteggia Fracture-Dislocation

This is one of the injuries around the elbow most frequently overlooked and at the same time most difficult to treat.

Mechanism.—This injury results from a fall against an object which acts as a wedge fracturing the upper third of the ulna and as the force carries on, the pronated radius is levered forward rupturing the annular ligament.

Post-Traumatic Anatomy.—This consists of a fracture of the upper third of the ulna with the fragments angulated forward associated with an anterior dislocation of head of radius with ruptured annular and radiohumeral ligaments. The biceps tendon tends to maintain this displacement.

Diagnosis.—This complicated injury should be suspected in all fractures of the upper third of the ulnar shaft. X-ray examination should be carried out to confirm the diagnosis.

The opposite displacements occur rarely when the violence is in reverse direction.

Treatment.—Early cases should be treated by operative reduction, internal fixation of the fractured ulna, and suture of the annular ligament. The elbow is splinted in flexion and supination for at least 3 to 4 months.

Late cases with nonunion require open reduction with bone grafting of the ulna and excision of the radial head. If malunion is present, resection of the head of the radius may be necessary to improve pronation and supination; reconstruction of the ulna is not usually indicated.

FRACTURES OF THE FOREARM

Fractures of the forearm may involve one or both bones and are caused by direct or indirect violence. The site of fracture may be in the upper, middle or lower third, the frequency increasing distally.

Mechanism.—A fall on the outstretched hand is the usual cause. When both bones are broken, the distal fragments are dorsally displaced. Direct violence generally gives a transverse fracture, and if both bones are broken, the fractures are usually at the same level.

Post-Traumatic Anatomy.—In the young, the greenstick type of fracture is common and considerable angulation is present. In the adult wide separation is possible.

In fracture of one bone, it must always be remembered that over-riding of the fractured ends can occur only with subluxation or dislocation of the other bone at the superior or inferior radioulnar joint. In fracture of the radius, muscular forces must be considered in analyzing the displacements. Fractures above the pronator teres have the upper fragment flexed and supinated by the biceps and supinator, while the lower fragment is pronated and displaced toward the ulna by the pronator teres and pronator quadratus. In fractures below the insertion of the pronator teres, the upper fragment is flexed and supinated to a minor degree, and

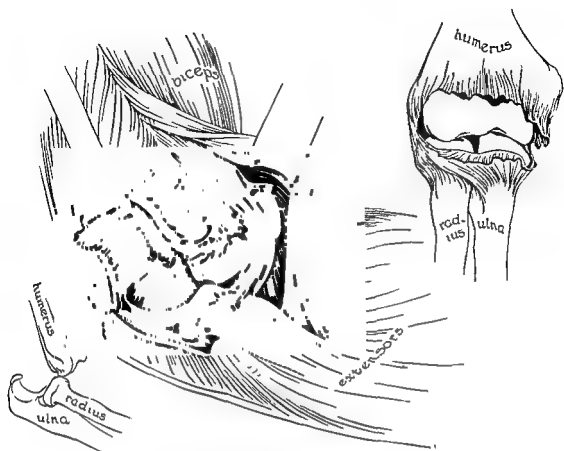


Fig. 358 —Posterior dislocation of elbow

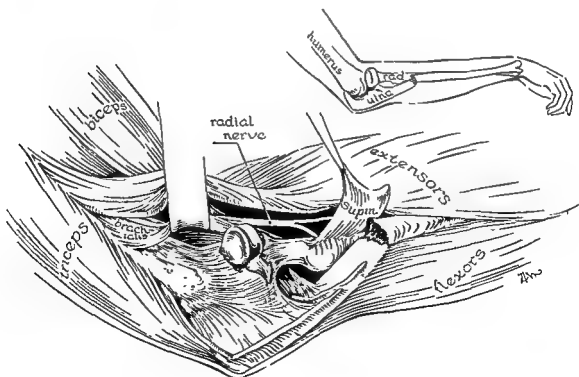


Fig. 359.—Monteggia fracture-dislocation.

Treatment.—From the point of view of therapy the fractures may be divided into:

1. Greenstick fractures.
2. Fractures without displacement.
3. Fractures with displacement.

Greenstick fractures are manipulated under general anesthesia by traction and direct pressure over the point of angulation. The angulation is just overcorrected. Great

tures of one or both bones without displacement are placed in the same type of cast. The time of immobilization varies from 6 to 12 weeks

Fractures with displacement are treated by:

1. Manipulation and plaster cast.
2. Open operation and internal fixation.
3. External skeletal fixation.



Fig 361.—Typical x-rays of fractured radius before and after plating.

care must be taken to avoid complete fracture and displacement.

A cast is applied from axilla to metacarpal heads with elbow at 90° , forearm in mid-position and wrist slightly dorsiflexed. Frac-

Manipulation under a general anesthetic with fluoroscopic or x-ray plate control is the safest method. Stable reduction must be obtained and a cast applied. The possibility of redisplacement must be checked

the lower fragment is displaced toward the ulna. In fractures in the lower third, the distal fragment of the radius is pronated and drawn toward the ulna by the pronator quadratus.

carefully reduced are usually stable. Fractures of one or both bones in the adult are prone to redisplacement after reduction unless the fractured surfaces solidly interlock. This tendency to instability after reduction

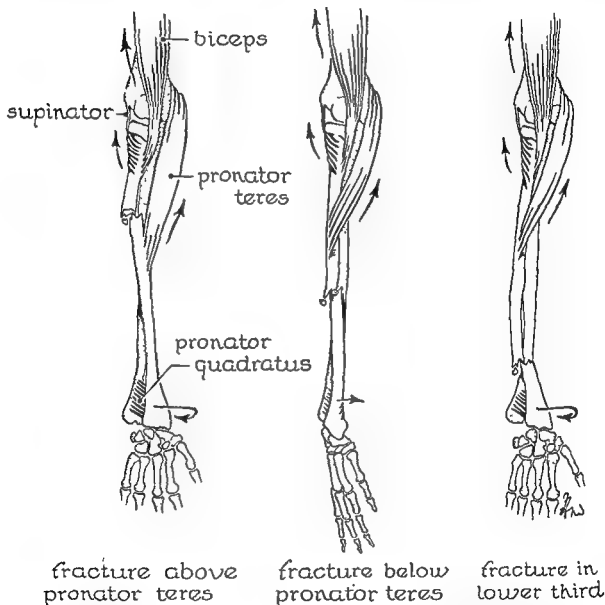


Fig. 360—Influence of muscle pull on fractures of the radius, at different levels

In injuries caused by direct violence, the direction of the force is the determining factor in the displacement.

The mechanics of the post-traumatic anatomy of forearm fractures is of the greatest importance. The greenstick fractures if

is the reason why fractures in this area are especially suitable for open operation and internal fixation

Diagnosis.—Diagnosis is readily made in cases of angulation or gross displacement. X-rays in two planes are essential.

reverse deformity with forward displacement is called Smith's fracture.

All cases, however, do not fall into this type and the variations are legion. It probably leads to poor work to classify all these cases as Colles' fractures and each must be analyzed and treated as an entity.

In the young adolescent, separation of the lower radial epiphysis, usually through the metaphysis, is the common injury. In older patients, a comminuted impacted fracture with foreshortening of the radius in relation to the ulna is common.

The most important factor in functional restoration is the mechanical alignment of the inferior radioulnar joint.

Fractures and epiphyseal separations with displacement are best reduced by the Hefser-rich or hand-shake method. The essentials are traction and countertraction for 3 to 5 minutes under general or local anesthesia. Following this disimpaction, the lower fragment is compressed volarward and toward the ulnar side. *It is almost impossible to overcorrect a Colles' fracture*, and adequate reduction of the triple displacement is essential. Both uncorrected displacements and foreshortening will give a derangement of the inferior radio-ulnar joint with interference to perfect rotation.

The essential point to ascertain after reduction is whether the reduction is stable.

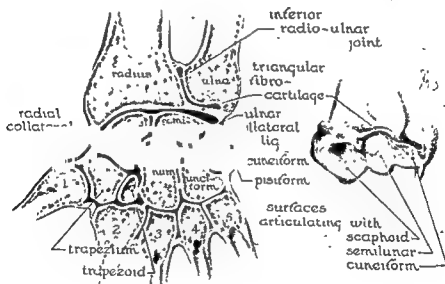


Fig. 363—Anatomy of the wrist and carpus

Derangement of this mechanism results in a limitation of pronation and supination, the latter being that more commonly limited.

Diagnosis is made on the typical deformity when present, the local tenderness, and radiological findings.

Treatment.—Fractures without displacement should be treated in a functional cast permitting all movements of the digits. Time factor 3 to 4 weeks.

The vast majority of correctly reduced fractures are stable and can be immobilized in a neutral position. (See General Section on Fractures) Probably not more than 15% are unstable as indicated by the tendency to immediate displacement after manipulation. These latter should be immobilized in volar flexion of 10° to 20° and ulnar deviation. The Cotton Loder position of full volar flexion is not recommended, because of the

by repeated x-ray. The cast is changed when the swelling subsides. Thumb traction associated with the cast may prevent some cases of recurrent displacement.

Open operation and fixation of the one bone in single fractures and both bones in dual fractures is the safest and best method in the hands of the specialist. Postoperative fixation in a cast is required for 2 to 5 months. Plates may be removed when union has occurred.

Longitudinal wire fixation has been used in fractures of these bones and is best for fractures of the ulna. External fixation by two pin units of the Roger Anderson or Stader type can be used by those working routinely with this method. Open operation with plating is advised in preference to this method.



Fig 362—X-ray of cross union.

Complications.—

Malunion is common in forearm fractures because of factors mentioned above together with the longer period required for consolidation in these bones.

Cross union may occur because of the tendency of the bones to approximate each other after fracture. This tendency is diminished by splinting in mid-pronation at which point the bones are farthest apart. Both malunion and cross union have their chief disability in limiting the range of pronation and supination. *Delayed* and *nonunion* are fairly frequent.

Dislocation of Superior Radioulnar Joint may persist after fracture of the upper third of the ulna in Monteggia fracture-dislocation.

Dislocation of Inferior Radioulnar Joint may result from fracture of the radius with over-riding or as a concurrent injury. If disabling, resection of the lower inch of the ulna gives considerable improvement in function.

FRACTURES OF THE WRIST

Fractures of the lower end of the radius, with or without injury to the articulating bones, constitute the largest group of cases attending traumatic clinics. These injuries occur at all age periods but are most frequent during the active period of life. In northern climates they increase during the winter months.

Mechanism.—The usual cause is a fall on the outstretched hand.

Post-Traumatic Anatomy.—The typical deformity described by Colles consists of three parts. The lower fragment is displaced (1) posteriorly, (2) radially, (3) with its articular surface rotated to face more dorsally.

If the radial displacement is sufficient, the ulnar styloid is avulsed with the ulnar collateral ligament. This triple displacement constitutes the dinner-fork deformity. The

tendency to circulatory interference. The typical functional cast should be applied. In the comminuted fractures and in unstable reductions, limitation of elbow movements by inclusion in the cast for 2 weeks is desirable. The average period for plaster splintage is 4 weeks, and during this period, active movements of the fingers are necessary. Elevation of the limb postoperatively to diminish swelling is required.

Complications.—

Malunion.—Many cases are seen where failure to secure adequate correction of the radial deviation has occurred. Most symptoms relate to the inferior radioulnar joint although many cases have function which is surprising in view of the deformity. Resection of the lower inch of the ulna will improve some of these cases.

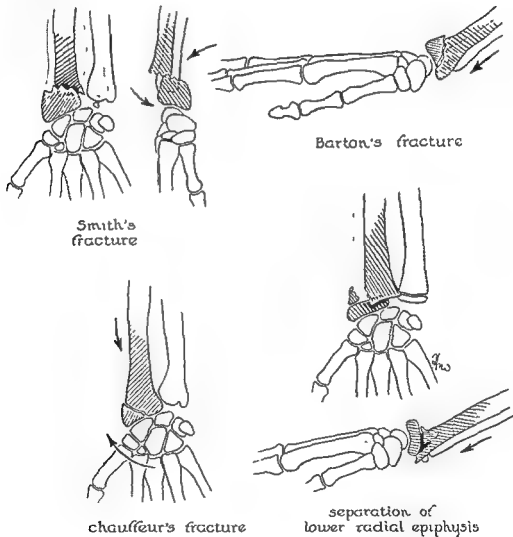
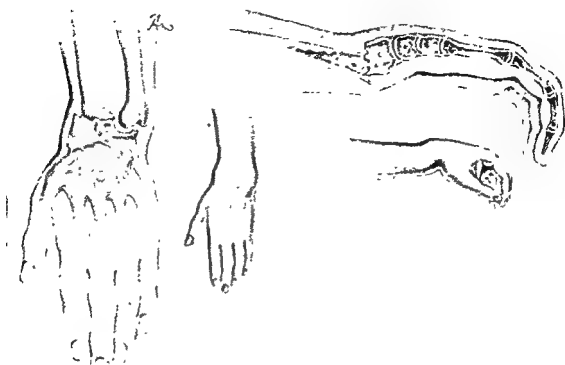


Fig. 365—Type fractures of lower end of radius

Reduction is successful up to 10 to 14 days. After this period refracture by closed or open procedures may be necessary in the younger patients.

After removal of the cast, exercises to restore pronation and supination as well as the function of the whole limb are indicated.

Sudeck's Atrophy.—Post-traumatic bone atrophy occurs most frequently after injuries at the wrist. With the earliest sign of painful swelling and stiffness of the fingers, the cast must be split, the limb elevated, and active movements of all fingers encouraged. This usually succeeds in arresting the vaso-



A



B

Fig 364—A and B Fracture of lower end of radius with posterior and lateral displacement (Colles' fracture)

fragment is displaced and this is often overlooked.

Diagnosis.—All sprains of the wrist with tenderness localized to the radial side of the joint and pain on dorsal or radial movements must be regarded as fractures of this bone until proved otherwise by radiological examination in anteroposterior, lateral, and especially oblique planes. If the x-ray is negative and the clinical symptoms and signs persist, x-rays must be repeated in 10 to 21 days. Often a narrow fracture line may be overlooked with inevitable nonunion.



Fig. 367.—Type fractures of the scaphoid in relation to the vascular supply.

to 6 months. Ischemic necrosis of the small ulnar fragment may be noted after 3 to 4 weeks by the relative density of this fragment in comparison to the radial fragment which is undergoing the decalcification of disuse.

Treatment.—Fractures of the tuberosity require only palliative therapy and give little disability.

All fresh fractures of the body require immobilization in a cast which places the wrist and thumb in the position of grasp. The thumb is best immobilized up to the interphalangeal joint. (See General Section on Fractures) Cases with displacement of the radial fragment need forcible manipulation through the full range of movement. The cast may be removed after six weeks and progress of union should be determined by x-ray. Some cases in young

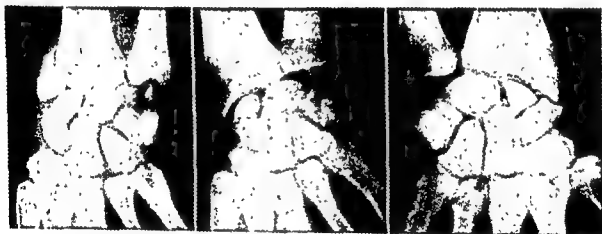


Fig 368—A X-ray showing the faint fracture line of fractured scaphoid associated with Colles' fracture. B. Aseptic necrosis of ulnar fragment C Nonunion of fractured scaphoid with arthritic changes.

Differential diagnosis from bipartite scaphoid is made by the even line between the fragments and the bilateral presence of this congenital anomaly.

The fracture line in the fresh case is usually narrow; after 10 to 21 days it appears broader because of the hyperemic decalcification. Cystic areas appear after 5 to 21 weeks and sclerosis may be present after 4

adults may join in 8 weeks but 16 weeks is more nearly the average. Union is determined when complete obliteration of the fracture line occurs.

If x-rays show ischemic necrosis of the ulnar fragment, two courses are open:

1. Continued plaster immobilization which may require 12 to 24 months.
2. Removal of the ulnar fragment.

motor disturbances. Should these measures fail, procaine injection of the inferior cervical ganglion should be considered.

Rupture of the Extensor Pollicis Longus Tendon may occur some weeks after the Colles' fracture from injury at the time of the fracture and subsequent attrition

Mechanism.—Fractures of the scaphoid are caused by falls on the outstretched hand with dorsiflexion and deviation to the radial or ulnar side. Fracture through the waist occurs when the two poles are locked and compressed. Forced ulnar deviation may cause a sprain fracture of the tuberosity.



Fig 366—X-ray showing the osteoporosis of Sudeck's atrophy with normal hand for comparison

FRACTURES OF THE SCAPHOID

Fractures of the carpal bones are not common. Injuries of the scaphoid are the most frequent and most important. Neglect of early recognition and immobilization leads to painful nonunion and traumatic arthritis.

The other carpal bones may be involved in sprain fractures or comminuted by direct or indirect violence. Such cases are rare and are discovered on radiological examination

Post-Traumatic Anatomy.—The common injury is a fracture through the waist. Sometimes the line of fracture passes more proximally giving a small ulnar fragment. The importance of this rests on the fact that the vascular supply to the scaphoid enters on a narrow area on the dorsal aspect of the bone and in the cases with a small ulnar fragment, this portion of the bone may be devoid of circulation, and undergo avascular necrosis. These are prone to nonunion. In certain cases of waist fractures, the radial

proximal part of the os magnum impinges on the posterior cornu of the lunate and ruptures the posterior radiolunate ligament. The lunate bone is then displaced forward rotating around the anterior radiolunate ligament. The carpus has now returned to its normal position and produced an *anterior dislocation of the lunate*. If the navicular remained in whole or in its ulnar part with the lunate, this bone would be displaced forward as well giving an *anterior dislocation of the lunate and navicular* or *anterior dislocation of the lunate and ulnar half of the navicular*.

best concentrated on the proximal articular surface of the os magnum which normally fits into the concave surface of the lunate. In such dislocations these articular surfaces are completely displaced from each other.

Treatment.—Reduction is achieved at the earliest moment under the full relaxation of general anesthesia. In the perilunar dislocations, reduction is easier than in the second stage. Long axis traction followed by direct pressure forward on the carpus while one finger firmly presses the semilunar backward from the volar aspect of the wrist re-

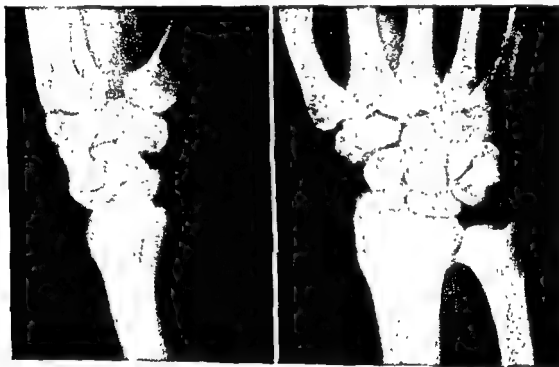


Fig 370.—X-ray of perilunar dislocation of the carpus

A chip fracture of the posterior cornu of the lunate may be present or a marginal fracture of the radius most commonly on its posterior rim. When the semilunar bone is dislocated anteriorly, pressure on the flexor tendons and median nerve is present.

Diagnosis.—The diagnosis may be suspected because of the dorsal displacement of the hand in the first stage or the anterior position of the semilunar on palpation in the second stage. Radiological examination is essential and in the lateral view attention is

sults in reduction and prevents this stage from being converted into the more difficult second stage.

In the anterior dislocation of the lunate, long axis traction with the hand gradually dorsiflexed is required to open up the space for the lunate. Direct pressure on the posterior pole in a distal and posterior direction will make this bone retrace its steps.

This dislocation may be reduced by closed methods up to 10 to 14 days; after this time open operation is usually required. If this is

There is no unanimity of opinion on this point and the case must be judged on economic and other bases. The conservative approach is the best in most cases and in clerical and professional personnel nonunion may be accepted and is often asymptomatic.

Cases of delayed or nonunion with pain interfering with work may be treated by drilling, bone graft, or arthrodesis of the wrist.

extension of the wrist. Complicated fracture dislocations result when the hand is caught in machinery.

Post-Traumatic Anatomy.—The forced hyperextension of the hand on the forearm causes the hand with the carpus to displace dorsally, leaving the lunate bone in its normal position. This is called a *perilunar dislocation of the carpus*. Sometimes the navicular bone remains beside the lunate giving

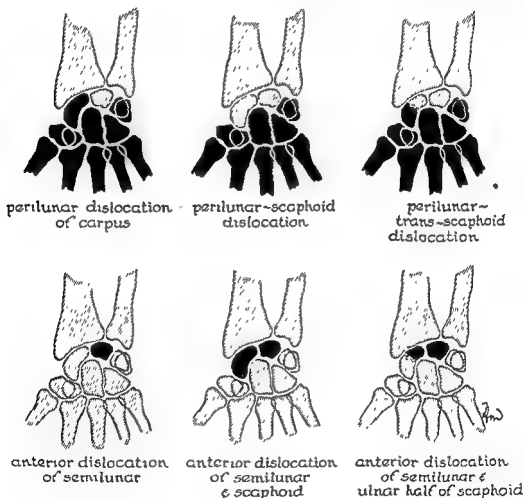


Fig. 369—Dislocations of the carpal bones.

DISLOCATIONS OF THE CARPUS

Dislocations of the carpus are uncommon and most occur in relation to the lunate and scaphoid bones, although carpometacarpal and other dislocations occur from unusual direct or indirect violence.

Mechanism.—The cause is usually a fall on the outstretched hand or forced hyper-

a *perilunar-navicular dislocation*. If a concomitant fracture of the navicular occurs, the ulnar fragment usually remains with the lunate, and the radial fragment displaces posteriorly with the carpus resulting in a *perilunar transnavicular dislocation*.

This is the first stage in the mechanism, since if forward recoil of the hand ensues, the



Fig 372—X-ray of fractured metacarpals.

stable reduction. The thumb should be splinted in a well-molded plaster cast for 3 weeks.

Fracture of the Neck of Fifth Metacarpal.

—In the usual case with considerable forward displacement of the head, reduction is obtained by flexing the metacarpophalangeal joint to a right angle and pushing forward on the shaft fragment while pushing back on the flexed finger. The fracture should be splinted to maintain position with these two opposing pressures.

Spiral Fractures of the Shafts are not usually seriously displaced. The hematoma on the palmar and dorsal aspects is a frequent complication and the discomfort is best relieved by elevation, repeated hot baths, and gentle active finger movements. Unless the fracture is very painful and requires protection from blows, no splint is required. When indicated a closely molded plaster cast can be applied which extends to the knuckles and leaves the fingers free.

Transverse Fractures of the Shafts.—Transverse fractures without displacement can often be treated by early gentle movements and hot baths to assist absorption of the hematoma.

Transverse fractures with displacement require manipulative correction and plaster

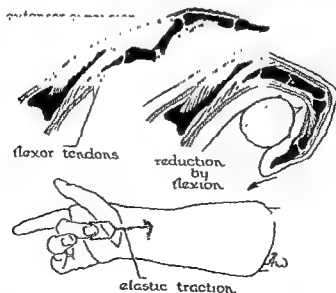


Fig 373—Splinting of fracture of proximal phalanx.

done within 3 to 4 weeks, the bones may be replaced; later than this the fragments may be devitalized and are probably best removed.

In dislocations without fracture of the scaphoid, immobilization in a functional cast for 3 weeks is sufficient. When a fractured navicular is present 12 to 16 weeks may be required and the time factor will be determined by x-ray evidence of union.

Most of these cases require a firm wrist strap protection when work is begun.

METACARPALS

Mechanism.—Fractures of the metacarpals are common and important injuries. They frequently result from a blow delivered by the fist or from a fall on the outstretched hand. Direct violence may be the responsible factor and crushing violence resulting in compound injuries are all too common among industrial accidents.



Fig 371.—X-ray of Bennett's fracture-dislocation

Post-Traumatic Anatomy.—When the force is directed along the adducted thumb as in boxing, the inner limb of the saddle-shaped base of the first metacarpal is sheared off against the greater multangular bone. This permits the carpometacarpal joint to dislocate outward and backward and results in the most common metacarpal injury, i.e., Bennett's fracture-dislocation (See Fig 311, G.)

Should the force be received directly on the flexed fifth finger and the knuckle, an impacted fracture of the neck of this metacarpal results. The head in this case is displaced forward on the neck and there is usually a noticeable deformity. This injury is second in frequency among metacarpal injuries.

The shafts of the metacarpals may be fractured in spiral or transverse manner and an important complication is the hematoma which frequently develops on the palmar and dorsal aspects of the hand.

Diagnosis.—Diagnosis of Bennett's fracture-dislocation is made by the painful swelling with deformity and crepitus in this area. It is to be differentiated from a transverse fracture of the base of the metacarpal which is not an intra-articular fracture and which is stable on reduction. X-rays will show the triangular medial fragment in the Bennett fracture-dislocation.

Local tenderness, posterior angulation in the shaft fractures and loss of the knuckle prominence in neck fractures are important clinical findings in other injuries.

Treatment.

Bennett's Fracture-Dislocation requires careful treatment to secure good results, and the instability of reduction must be overcome by two methods:

1. Long axis traction in moderate abduction.
2. Direct pressure medially on the base of the metacarpal.

Traction and splintage are required for four weeks. Failure to carry out this treatment results in enlargement of the joint, with pain and development of traumatic arthritis.

Fracture of the Base of First Metacarpal is usually impacted with the distal fragments in the adducted position. Direct pressure over the external angulated prominence associated with long axis traction produces a

capsule while the base of the phalanx is displaced posteriorly. Such dislocations most frequently occur in the thumb, and the head of the metacarpal is buttonholed on each side by the two heads of flexor pollicis brevis.

side to side while exerting lateral and posterior pressure on the metacarpal head.

If closed reduction fails, dorsolateral incision is made and the buttonhole opened to permit reduction.

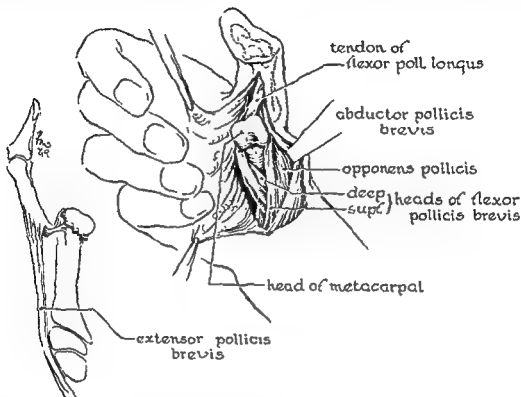


Fig 375.—Backward dislocation of thumb.

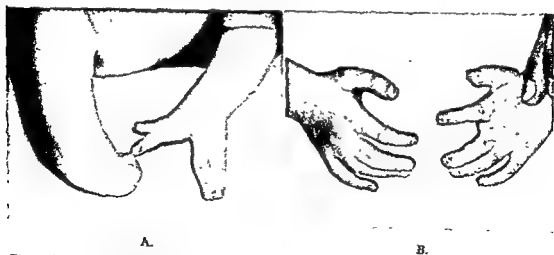


Fig 376—Congenital deformities. A. Absence of right hand and failure of differentiation of fingers of left hand. B. Syndactylism of long and ring fingers.

Reduction.—Reduction is usually possible by hyperextending the thumb and while maintaining traction, drawing the digit from

The joint should be splinted for 7 to 10 days in a flexed position. This is followed by gentle active movement.

splintage. Incorporation of traction on the corresponding flexed fingers may be required to maintain position. When closed manipulation fails, open operative correction without internal fixation can be safely advised. Only a small dorsal incision is required to permit replacement and interlocking of the fragments.

Fractures of the Phalanges.—Fracture of the proximal phalanx is that which gives most difficulty. The interossei and lumbricals attached to the dorsal aponeurosis buckle the fragments so that the distal fragment is hyperextended and the proximal fragment flexed. Treatment consists in manipulation of the fragments into the correct alignment and maintenance with the finger flexed and long axis traction arranged to line up with the scaphoid tuberosity. Three weeks of elastic traction are required during which period the other digits are constantly and actively exercised.

reflection will suffice. No anesthetic is required. The nail is left as a protection to the lacerated bed unless practically avulsed.

Mallet or Dropped Finger.—Mallet finger results when the terminal phalanx is suddenly flexed while the extensor mechanism is taut. It frequently occurs in baseball and cricket players but the housewife may present the deformity after stubbing the digit. The extensor tendon may be torn off the base of the distal phalanx with or without its bony insertion.

Treatment to be effective must be carefully followed and may be one of two types.

1. Splinting in a plaster cast without interruption for six weeks with the distal joint hyperextended and middle joint flexed.

2. Suture with removable suture placed through the phalanx.

If either of these methods from the outset appears impractical, the hand may be per-



Fig 374—Mallet finger.

Crush Injury of Distal Phalanx is one of the most common hand injuries. Most cases are compound fractures with severe damage to the soft parts.

In the average case, careful cleansing in saline solution followed by local application of a penicillin dressing and splinting will suffice. The minimal use of sutures is advocated. If possible the nail is left as a splint.

Subungual Hematoma.—If the nail is elevated from its matrix, evacuation of the blood by a lateral incision at the cuticular

mitted full movements with protection from strain or injury and will heal with some flexion deformity and stiffness of this joint. This is all too frequently the end result from failure to splint in the correct position for the time indicated.

Dislocations

Metacarpophalangeal Dislocations

These most frequently are caused by violent hyperextension. The head of the metacarpal is forced through the front of the

Phalangeal Dislocations

These may occur in any direction but usually the distal phalanx is displaced posteriorly. Reduction is achieved by traction and opposing pressure on the two phalanges. The joint should be splinted in flexion for 7 to 10 days.

THE HAND

The hand is the effector organ of the upper extremity and is highly specialized for the function of grasp. The mobility of the fingers is essential for its proper use.

Congenital Anomalies

Developmental anomalies may occur at the wrist, in the hand proper, or in the fingers. The possibilities are legion.

The hand may present a clubbed deformity as in the foot, deviating to the ulnar or radial side. Madelung's deformity results from a derangement of the lower radial epiphysis with overgrowth of the ulna. The ulna projects dorsally and the whole hand is displaced in a volar and radial direction.

Lobster hand occurs with failure of development of the medial side of the hand and the four digits.

There may be any combination of deformities in the fingers with a variation in number, length, or size. Fingers may be partially or completely differentiated. Syndactylism is a common defect.

TENDONS

The hand depends for its function on the mobility of its small joints and on the proper mechanism of the tendons arising in the forearm and the intrinsic muscles of the thenar and hypothenar eminences together with the lumbricals and interossei.

The movements of the hand and especially of the fingers require the preservation of the smooth gliding surfaces of the tendons in their sheaths which injury, infection, and inadequate surgery tend to destroy. No field of surgery requires greater understand-

ing, constant study, and meticulous technique than does the care of hand injuries. The work of Bunnell, Koch, Mason and Allen, together with the Hand Centers in the United States during the past war has been the chief force in raising the standard of therapy in this sphere.

Healing of Tendons.—The healing of cut tendons can be correlated with that in fractures since in both, there is laid down a temporary scaffold, which is followed by the permanent union. This temporary tissue represents the connective tissue proliferation from the paratenon, epitenon, endotenon and tendon sheath. After tendon severance, a jelly-like substance extends from the cut ends into which the connective tissue grows for about half an inch along each and there is increased vascularity and swelling. *During the first week* a fusiform enlargement develops, which increases throughout the second week. If the tendon is confined within a thecal tunnel during this period, the swelling may result in ischemic necrosis and eventual dense adhesions.

The tendon cells begin proliferation in the second week. There is little strength in the union until the third week when the tendon fibers begin to bridge the gap.

Between the third and fourth week the healing mass begins to separate from the surrounding tissues and the fusiform swelling contracts and increases in tensile strength. This is the stage and time when graduated function increases the speed of union as well as the re-establishment of the gliding mechanism.

Mechanism of Injury.—The tendons may be individually severed by the puncture wound of a sharp spicule of glass or metal. Several may be sectioned with associated injury to nerves, blood vessels, bones and joints in machinery, or by lacerations on knives, broken bottles, china or through glass windows.

Diagnosis.—A most careful history and examination of the movements of the hand



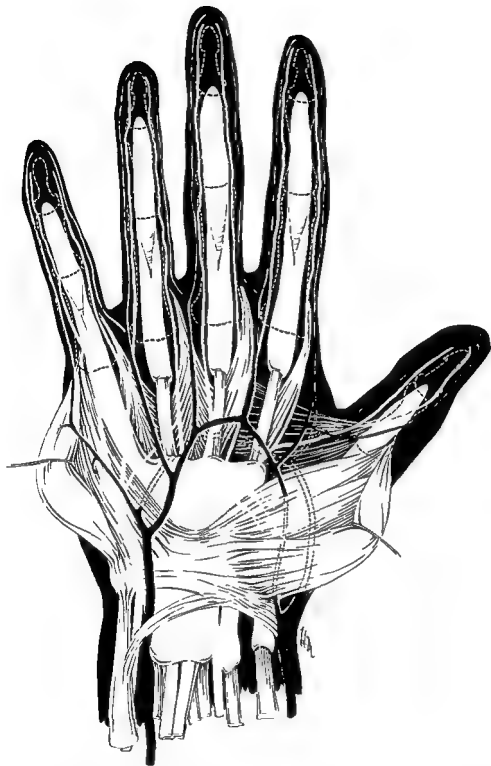


Fig 377A —Anatomy of the palmar aspect of the hand

The arterial circulation, in red, shows the communication between the radial and ulnar arteries to form the superficial palmar arch and its digital branches, and, in dotted lines, the formation of the deep palmar arch and its anastomoses

The ulnar and median nerves, in yellow, supply the digital sensory branches to the ulnar $1\frac{1}{2}$ and radial $3\frac{1}{2}$ fingers respectively

The digital, ulnar, and radial synovial sheaths are also shown



Fig 377B—Anatomy of the dorsal aspect of the hand

The dorsal arterial circulation, in red, is shown with the radial artery terminating by passing between the two heads of the first dorsal interosseus muscle

The digital sensory supply, in yellow, from the radial and ulnar nerves is shown Also the various extensor synovial sheaths in their relation to the dorsal carpal ligament.



and individual tendon functions must be made. Concomitant injuries to digital or major nerves must be determined by attention to sensory and motor loss. X-rays should be taken to ascertain the extent of skeletal damage.

Any defect in the movements of the fingers should suggest a cut tendon, and this must be excluded when the wound is cleansed and sutured.

of complications is greatly increased. Antibiotics should supplement the most careful wound toilet, and sepsis should be avoided at all costs. *Tendon injuries are acute emergencies and should be so regarded from the moment when first seen by the medical profession.*

Antitetanic serum should be given as a routine. If previously immunized, a booster dose of toxoid can be prescribed.

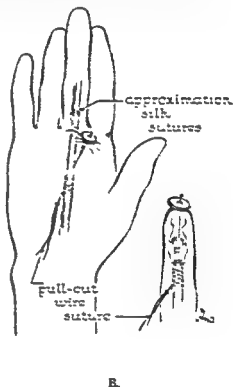
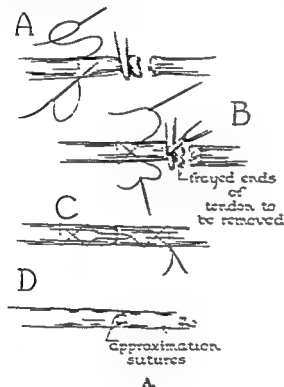


Fig. 378.—A. The silk technique. B. Bunnell technique.

Principles of Treatment.—The treatment of cut tendons is determined by the environment in which the injury occurs. If near a center where adequate facilities exist, the case should be immediately referred. Tendon suture is not a procedure for the general practitioner. It is best, if specialist facilities are unavailable, to carry out a careful cleansing of the wound and skin suture and then refer the patient later for secondary tendon repair. This will result in much better results than if primary suture is attempted with inadequate technique.

The time factor is of the greatest importance. After 2 to 3 hours the development

Technique of Suture.—There are two methods of tendon suture to be considered:

1. The silk technique.
2. The removable suture of stainless steel (Bunnell).

Both have their place and both require experience to obtain good results.

Principles Underlying Operative Treatment.—

1. The most delicate handling of the tissues is required.
2. If both flexor tendons are sectioned in "No Man's Land," i.e., between the distal palmar and distal digital crease, the sheath

should be exposed in its length and removed except for retaining bands over the joints. The flexor sublimis should be routinely removed if cut proximal to its insertion and only the profundus sutured.

3 In secondary suture of the flexor tendons, removal of the sublimis and profundus and replacement with a tendon graft from the palmaris longus sutured in the palm to the profundus tendon and in the theca at the base of the distal phalanx, give the best results

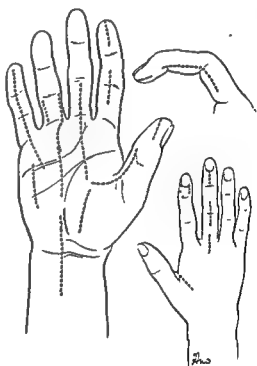
vation and by evenly applied compression dressings, with avoidance of constricting bandages.

3. The best physiotherapy is the voluntary movement of the fingers in warm soapy water. Lanoline is useful to soften hardened skin. Sponge rubber can be used for grasp exercises.

Results.—The results of tendon suture are greatly improved by attention to the above principles. When cut flexor tendons in the fingers have been properly repaired, the tip



A. correct



B. incorrect

Fig. 379—Lines of incision (after Bunnell).

4 Incisions for the fingers should be in the midlateral plane, never longitudinally over the volar surface.

5. In the palm, incisions should follow the flexion creases.

Principles Underlying Postoperative Therapy.—

1. Immobilization with minimal tension is required for 3 weeks for the flexor and 4 weeks for the extensor tendons.

2. Every effort must be made to avoid postoperative swelling of the hand by ele-

vation of the finger can be flexed to the distal palmar crease. This is also the result to be expected after tendon graft has been employed in place of secondary suture. Repair of the cut extensor gives on the average a much better functional result than that of the flexor tendon.

INFECTION OF THE HAND

The hand is subject to the same infective processes as are the corresponding tissues in other regions of the body. The hand, how-

ever, is especially prone to involvement because of its functional activities.

Historical.—The work of Kanavel and his followers did much to improve our understanding of this subject. The knowledge of the tremendous morbidity resulting from hand infections together with the better understanding of the value of prevention and accurate therapy has done much to lessen the ravages heretofore so frequently seen. The rise of industrial medicine with doctors and nurses present in the large factories enables prophylactic treatment of minor injuries and rapid transfer of the major accidents to specialist facilities. Finally, the development of the sulfonamides and now the antibiotics have given the profession powerful agents to minimize hand infections. The future control rests in prophylactic care at the time of injury and the use of the chemotherapeutic agents at the earliest moment associated with the most accurate and conservative surgery of this most important part of the body. Already the influence of these principles is obvious to those who compare this problem in large hospitals today with the period prior to the last war.

Infections of the Fingers

Felon

This type of infection is peculiar to the hand and connotes a cellulitis of the pulp space. The cause is usually a prick of the volar surface. Because of the fascial septa limiting swelling, tension rapidly develops with acute throbbing pain. This pain is

especially severe if the hand is dependent, and is relieved by elevation. If the tension is allowed to develop beyond a certain point the circulation to the distal phalanx is occluded with necrosis and subsequent osteomyelitis.

Diagnosis.—The diagnosis is made on the history of rapidly increasing pain, localized to the pulp space. The tense swelling of the area is noted on palpation. The point of injury may or may not be visible.

Therapy.—Elevation and immediate penicillin therapy will clear the early cases. If seen late, an incision on the side sectioning the septa should be performed.

Paronychia

This is defined as an infection around the nail. The bacteria enter through a crack on the thickened cuticle of the manual worker or through a hangnail in the office employee. In hospitals, this infection was frequent in nurses when the nail brushes were not sterilized. One of the common causes today is the injury during manicure with nail file or clipper.

The process begins superficially as a subcuticular infection at the side of the nail. This forms a localized bead of pus, which evacuates itself or is easily evacuated. The infection may spread under the nail at the side and develop into a subungual abscess lifting the nail from its bed. This is especially severe if the spread is to the base of the nail which is raised from its matrix. The skin overlying the nail base is raised, swollen, reddish or bluish in color. The infection may spread completely around the nail, the so-called "runaround."

Diagnosis.—The clinical appearance at the different stages makes the diagnosis. Transillumination will show the pus beneath the nail as an opaque area.

Treatment.—Bathing the finger in hot water will soften the skin and accelerate localization and evacuation. When pus is present a small incision without anesthetic frequently suffices.

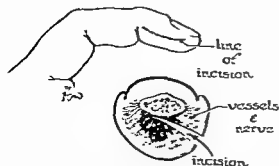


Fig. 320.—Incision of felon.

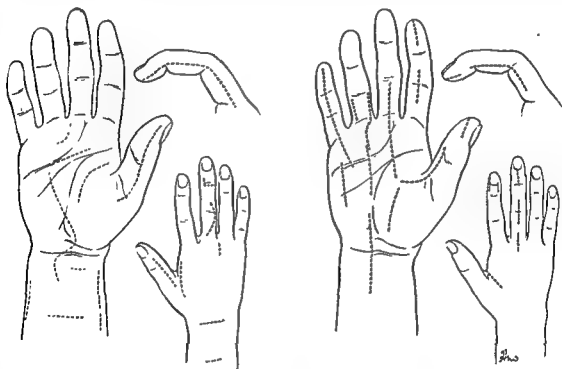
should be exposed in its length and removed except for retaining bands over the joints. The flexor sublimis should be routinely removed if cut proximal to its insertion and only the profundus sutured.

3 In secondary suture of the flexor tendons, removal of the sublimis and profundus and replacement with a tendon graft from the palmaris longus sutured in the palm to the profundus tendon and in the theca at the base of the distal phalanx, give the best results

vation and by evenly applied compression dressings, with avoidance of constricting bandages.

3 The best physiotherapy is the voluntary movement of the fingers in warm soapy water. Lanoline is useful to soften hardened skin. Sponge rubber can be used for grasp exercises

Results.—The results of tendon suture are greatly improved by attention to the above principles. When cut flexor tendons in the fingers have been properly repaired, the tip



A correct

B incorrect

Fig 379.—Lines of incision (after Bunnell).

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Principles Underlying Postoperative Therapy.—

1. Immobilization with minimal tension is required for 3 weeks for the flexor and 4 weeks for the extensor tendons.

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vation of the finger can be flexed to the distal palmar crease. This is also the result to be expected after tendon graft has been employed in place of secondary suture. Repair of the cut extensor gives on the average a much better functional result than that of the flexor tendon.

INFECTION OF THE HAND

The hand is subject to the same infective processes as are the corresponding tissues in other regions of the body. The hand, how-

contains the tendons to the index and pollex together with the thenar musculature.

The middle palmar space thus exists deep to the flexor tendons and extends distally along the arcular spaces around the lumbrical muscles to the three web spaces. Proximally it extends through the carpal tunnel into the forearm deep to the flexor tendons.

The thenar space extends distally along the lumbrical canal to the radial side of the index finger.

The importance of these fascial planes rests in the fact that they determine the passage taken by pus in its extension.

Dorsal Spaces

The dorsal subcutaneous space is found superficial to the dense layer of fascia and aponeurosis joining the extensor tendons. Deep to this plane is the subaponeurotic space. Pus contained in the deep space usually points at the periphery of the aponeurotic layer but may point directly dorsally in a collar-stud type of abscess.

Paths of Infection.—The middle and thenar spaces are those of the greatest surgical importance. Infection in the middle palmar space arises from direct puncture or lacerated wounds or from extension of deep infection of the long and ring fingers. Suppurative tenosynovitis of these two fingers or web infection may afford the source of infection by extension proximally.

The thenar space is involved by extension from deep infections of the index and volar aspects of the thumb.

The dorsal subaponeurotic space may be involved directly by puncture or lacerated wounds or secondarily by extension from the volar infections. Lymphatic drainage of the fingers extends to the dorsum and may carry the bacteria to the dorsal subcutaneous or subaponeurotic spaces.

Clinical Picture.—In severe infections the general reaction to infection will be present with raised temperature, pulse rate, and malaise.

Local signs give evidence of the infected space and vary with the extent and intensity of the inflammation.

Infection of the middle palmar space results in a swollen, brawny induration of the palm; the fingers are flexed, and often, too, the wrist. Attempts to extend the fingers are resisted because of pain. Tenderness is localized over the middle of the palm. The site of the entrance of the bacteria may be seen. Because of the lymphatic drainage to the dorsum there is considerable swelling of this area as well.

Infection of the thenar space presents greater swelling due to the absence of the resistance of the palmar fascia. The tension is minimized by the abducted position of the thumb. The swelling is marked both on the palmar and dorsal surfaces.

Infections of the dorsal spaces present a reddened and convex surface. The appearance is similar in the swollen dorsum seen in extension of edema from the palmar infections. The differential diagnosis is important and is made by a study of the path of infection and by the presence of fluctuation.

Treatment.—*Prophylactic.*—Fascial space infections are best prevented by appropriate care of initial injuries and the earliest possible use of penicillin when infection has developed.

Curative.—The hand should be carefully wrapped in bulky dressings which also afford splinting in the functional position of the hand and wrist. The part should be elevated. If the infection is severe with general manifestation, the patient is best treated in bed. Massive penicillin therapy should be given. When the process is localized and pus is present, surgical drainage is carried out. Local penicillin may be instilled. A culture of the pus and a study of its penicillin sensitivity are important.

SUPPURATIVE TENOSYNOVITIS

Infection of the synovial sheaths of the flexor tendons prior to the use of penicillin was a most crippling disease. Few cases

When the spread has occurred under the base of the nail, the nail should be transected at the lanula and the proximal portion avulsed under general or regional anesthesia. Local and systemic penicillin will rapidly clear the infection.

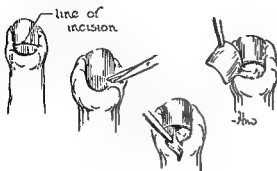


Fig 381—Drainage of paronychia and subungual abscess

Web Infection

One of the three triangular web spaces of the fingers may be infected by a prick, cut or crack in the overlying dermis. The infection usually localizes forming a pocket of pus, which tends to point dorsally. The process sometimes extends into the hand and involves the middle or thenar space.

Diagnosis.—The reddened swollen area causing the adjoining fingers to spread and with the swelling spreading onto the dorsum of the fingers is characteristic.

Treatment.—The hand should be elevated and when the patient is ambulatory this can be secured in a sling. Hot bathing gives relief of pain. Systemic penicillin is administered routinely. Incision will be required when the pus is localized.

FASCIAL SPACE INFECTIONS

Surgical Anatomy.—Three palmar and two dorsal fascial spaces should be considered.

- Palmar: Middle palmar space.
- Thenar space.
- Hypothenar space.
- Dorsal: Subcutaneous
- Subaponeurotic.

Palmar Spaces

The palmar fascia forms a dense roof overlying the deep structures of the palm, consisting of the flexor tendons, the branches of the median and ulnar nerves and the vessels. The floor is formed by the metacarpals with the fascia overlying the interossei. From the ulnar border of the palmar fascia, a septum passes to be attached to the fifth metacarpal and separates the *hypothenar space* muscles from the *middle palmar space*. On the radial side, a membranous septum passes to the third metacarpal forming the lateral boundary of the middle palmar space, which is thus separated from the *thenar space*. This latter consists of the area on the volar surface of the adductor pollicis and

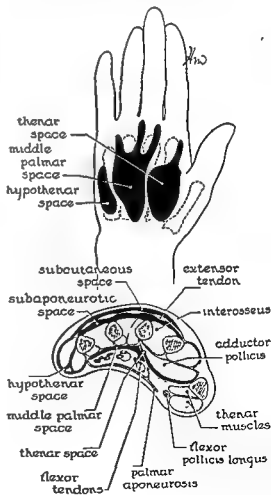


Fig 382—Fascial spaces.

ulnar bursa is involved. Attempts to extend the fingers give rise to intense pain.

Treatment.—*Prophylactic.*—Immediate care should be given to puncture wounds. In medical personnel this is important in needle or scalpel pricks on septic cases. Careful sucking out of the blood and washing to encourage bleeding followed by soaking in alcohol is desirable.

Penicillin should be given at the earliest sign of infection.

Curative.—Bed rest is indicated when infection has developed and the part should be splinted and elevated. Systemic penicillin should be forced.

The tendon sheath is best opened under tourniquet and general anesthesia, when the diagnosis is established. Local penicillin should be instilled through a fine catheter.

When one digital sheath alone is involved and is seen late, amputation must be considered with the exception of cases involving the thumb. This is especially true if involvement of bone or joint is present.

LYMPHANGITIS

Lymphangitis is the most fulminating infection found in the hand. From a small prick a streptococcus or staphylococcus strain may enter the lymphatic system and rapidly spread proximally along the lymphatic channels.

The red streaks up the inflamed lymphatics are readily seen and this renders diagnosis easy.

Treatment.—When first suspected, the patient should be treated in bed. In severe infections, general therapy directed to maintain the fluid balance is important. Today with penicillin this dread process can rapidly be brought under control. Systemic penicillin is forced. The limb is elevated and splinted. Cooling lead and opium or hypertonic saline dressings may be applied to diminish pain.

HUMAN TOOTH WOUNDS

Human tooth wounds of the hand are serious injuries and result from bites or a blow

by the fist on the teeth. The wounds usually involve the knuckle area and penetrate through the extensor tendons into the adjacent joints.

Bacteriology.—*Streptococcus*, *staphylococcus*, *Bacillus fusiformis*, and spirochetes of Vincent's angina, *E. coli*, proteus and anaerobic streptococci.

Clinical Course.—Unless treated early, the process runs a rapidly progressive course with the development of a foul-smelling wound from involvement of tendons, joint, and subaponeurotic planes.

Treatment.—These wounds should be most seriously considered as the organisms are acclimated to human tissues. Surgical débridement is required with careful cleansing with saline, peroxide, and finally penicillin instillation. The wounds are best left open and subjected later to secondary suture.

When seen later the path of infection should be opened, and the wound treated with zinc peroxide. Cultures should be taken to indicate whether penicillin or streptomycin would be more efficacious.

DUPUYTREN'S CONTRACTURE

This consists of a contracture of the palmar fascia together with its digital extensions. It is named after Dupuytren, who described the pathology and clinical findings, after dissecting a hand involved with this disease.

Clinical Picture.—The process is more common in the male sex and contrary to common opinion, in the sedentary than in the manual worker. There is a hereditary tendency and it is prone to occur in families subject to gout and cardiovascular degeneration. It has appeared acutely after coronary thrombosis. The patients are usually those with close-knit joints with a tendency of stiffness thereof even after minor trauma. The contracture develops in later years but has been seen in adolescence.

The thickening first appears deep to the distal palmar crease and extends into the ring finger. The fingers involved in order of

indeed preserved the mobility of the fingers and hand. Today the cases are less frequently seen and when treated by prompt incision and local and systemic penicillin, functional restoration can frequently be expected.

Surgical Anatomy.—The flexor tendons of the thumb and fingers are surrounded by a synovial sheath. In the case of the thumb and little finger, this sheath extends from its insertion proximally into the radial and ulnar bursae surrounding the flexor pollicis longus and the flexor sublimis and profundus tendons, respectively. The synovial covering on the tendons of the index, middle, and ring fingers extends from the insertions to the distal palmar crease.

infection occurs on the thumb or little finger, involving the synovial sheath, extension will occur to the radial or ulnar bursa or finally both with possible rupture into the forearm fascial space.

On the index finger, extension proximally will be into the thenar space, and on the long and ring fingers, into the middle palmar space.

Paths of Infection.—The synovial tendon sheath is usually involved from a puncture wound or laceration in the flexion creases on the digits. This may be a micro- or macroscopic wound.

Clinical Picture.—The symptoms and signs of suppurative tenosynovitis are more marked than those of fascial space infections

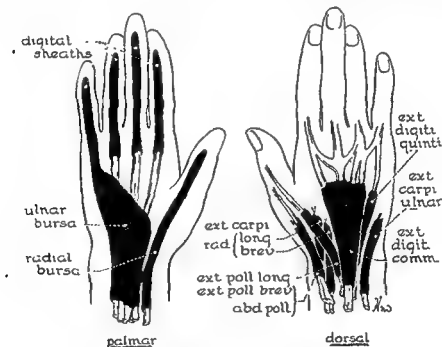


Fig 383—Synovial sheaths.

The radial and ulnar bursae lie close together in the carpal tunnel deep to the annular ligament. Communication exists here in the majority of cases. The sheaths extend proximally for a short distance above the annular ligament, projecting into the forearm fascial space.

The course of extension of the infective process can thus be readily seen. If the

Toxic absorption is rapid and the patient looks and feels ill with raised temperature (102° to 104°) and pulse rapid.

Locally there is exquisite tenderness localized to the digital sheath or to the ulnar and radial bursae, where extension has occurred. The finger is flexed and swollen when a single sheath is involved or the whole hand is flexed and swollen when the

Pathology.—Various theories exist as to their nature. Some believe them to be of neoplastic origin with the formation of the cystic contents by secretion of the connective tissue cells. Others hold that they develop because of connective tissue irritation in an area of mechanical derangement where ligament or tendon sheath plays over a bony prominence. Ganglia frequently communicate with joints, but few are due to herniation of the synovial lining.

Treatment.—Many ganglia around the wrist disappear spontaneously if a wrist support is employed. This is especially true of those over the dorsal aspect of the lunate bone.

The ganglia can be evacuated by direct blow or needling. The best method to secure a cure is by careful excision.

Epidermoid Cysts

Cystic swellings are found on the hands of manual workers and follow wounds with implantation of epithelial cells. Hence they are frequently called implantation dermoids. The cysts contain epithelial debris rich in cholesterol.

Treatment.—Excision.

Trigger Finger

This is an interesting anomaly which results from a thickening in the proximal end of the tendon sheath near the distal palmar crease or a swelling of the tendon mass where the sublimis divides to cross the flexor profundus. This results in a locking of the tendons at this point in the sheath, whereupon if the patient forcibly extends, the finger suddenly moves with a jerk.

Treatment.—Under local anesthetic, the area is exposed through a transverse incision. The sheath is sectioned at the side for approximately one inch, whereupon movements of the finger demonstrate that the mechanical derangement has been relieved.

Tendovaginitis of the Radial Styloid (DeQuervain)

This condition should be correlated with trigger finger. It consists of a thickening of the tendon sheath over the abductor pollicis longus near the radial styloid. Mechanical irritation is probably the cause.

The patient complains of pain radiating from this area over the extensor aspect of the thumb and up the forearm. The lateral aspect of the lower end of the radius may show a tender fusiform swelling.

Treatment.—Under local anesthesia a transverse skin incision and longitudinal section of the tendon sheath are made. Immediate function is permitted. The results are excellent from this opening of the sheath.

Tuberculous Tenosynovitis

The synovial sheaths of the flexor and extensor tendons may be involved by tuberculosis. This is frequently secondary to disease elsewhere such as in the lungs or lymphatic glands. Characteristically the ulnar bursa is involved with the formation of a cystic swelling extending under the carpal ligaments in dumbbell-shaped arrangement. Fluctuation can be obtained from the forearm to the palmar portions, and the condition is referred to as a *compound palmar ganglion*. The cyst may contain fibrinous bodies of the melon seed or rice body variety.

Treatment.—The treatment is both general and local. The patient must be regarded as one with systemic tuberculosis and the treatment of the hand is incidental and should be timed for a period when the systemic reaction is minimal. Complete excision of the involved tissues is the ideal surgical therapy. Streptomycin should be the associated chemotherapy.

REFERENCES

- Bunnell, Sterling: *Surgery of the Hand*, ed. 2, Philadelphia, 1947, J. B. Lippincott Company.

frequency are the ring finger, little finger, middle finger, index and thumb. The condition is frequently bilateral. There is an associated tendency to contracture in the plantar fascia and also in the fascia between the corpus spongiosum and corpora cavernosa of the penis.

Pathology.—The process begins as a fibroblastic proliferation in the palmar fascia deep to the distal palmar crease and on the ulnar side of the hand. In the early stages there are signs of an active inflammatory process with round cell infiltration. The process runs a variable course, varying in its

vised to avoid chronic trauma such as pressure or strain to the involved tissues for although trauma probably does not initiate the process, it appears to aggravate it. The use of a chamois glove for work or sports is helpful.

Operative therapy should be considered before the skin becomes too involved and the finger joints stiffened. If seen early, complete excision of the palmar fascia and its extensions through incisions in the palmar creases is best. If the skin is grossly involved, it must be removed and replaced by a split thickness skin graft. When marked

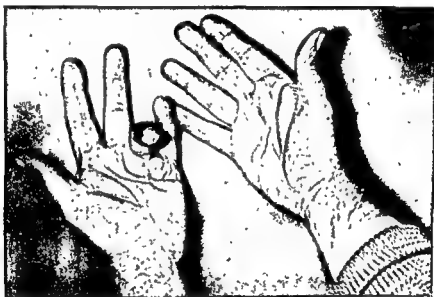


Fig 384—Dupuytren's contracture

extent and intensity. Gradually, however, the fibrosis involves the skin with loss of the fat lobules and skin glands by pressure atrophy. The extension to the fingers causes flexion of the proximal joints and increasing stiffness. The ring and other fingers are curled into the palm and are awkward when working and embarrassing when shaking hands.

Treatment.—Each case must be judged according to the stage of the disease and the actual disability to the individual patient.

When seen early the patient should be ad-

vised to avoid chronic trauma such as pressure or strain to the involved tissues for although trauma probably does not initiate the process, it appears to aggravate it. The use of multiple subcutaneous fasciotomies does not give good results but may be employed as a preliminary to radical excision in severe cases.

GANGLIA

Ganglia are cystic swellings which are found in many parts of the body arising from joint capsules, ligaments and tendon sheaths. They are especially common on the hands and feet.

CHAPTER XXXIX

FRACTURES AND OTHER DISORDERS OF THE LOWER EXTREMITY

H. F. MOSELEY, D.M.

PELVIS

Fractures of the pelvis are serious injuries and are frequently complicated by associated trauma to the urethra, bladder, and rectum. In this respect they are comparable to fractures of the skull and spine where involvement of the brain, cord, and meninges constitutes the major problem in therapy.

Mechanism of Injury.—Fractures of the pelvic ring are caused by lateral or antero-posterior compression. The accidents of travel in train, car, and plane account for some. Crushing beneath heavy weights or between cars account for others. Direct falls on the trochanteric area may fracture the acetabulum. Violence to different areas causes isolated fractures, e.g., the ilium.

Classification.—

1. Pelvic Ring.

- (a) Superior and/or inferior pubic ramus, unilateral or bilateral.

- (b) Diastasis of symphysis pubis.

- (c) Associated with (a) or (b), fracture through ilium or sacrum on same or opposite side or sacroiliac dislocation.

2. Acetabulum

- (a) Rim. With posterior or anterior dislocations of the hip.
- (b) Floor. With varying degrees of central dislocation of the hip.

3. Isolated Fractures.

- (a) Avulsion—

- (i) Anterior inferior spine by rectus femoris.
- (ii) Anterior superior spine by sartorius.
- (iii) Ischial epiphysis by hamstrings.

- (b) Direct trauma. Wing of ilium.

4. Sacrum and coccyx.



diastasis
of symphysis



fracture
through pubis



fracture of pubis
of sup & inf. rami
on opposite sides



ant & post fracture
with upward
displacement



post. dislocation with
fracture of post. rim
of acetabulum



central dislocation
of hip

Fig 385—Type fractures of the pelvis

- Codman, E. A.: *The Shoulder*, Boston, 1934, The Author
- Couch, John Harold: *Surgery of the Hand, Some Practical Aspects*, Toronto, 1939, University of Toronto Press
- Cutler, Condict W.: *The Hand, Its Disabilities and Diseases*, Philadelphia, 1942, W. B. Saunders Company
- De Palma, A. F.: *Surgery of the Shoulder*, Philadelphia, 1950, J. B. Lippincott Company
- Gordon, Ian: Expectant Treatment of Pyogenic Infections of the Hand With Special Reference to Infection of the Flexor Aspect of the Fingers, *Brit J Surg* 38: 331-339, 1951
- Handfield-Jones, R. M.: *Surgery of the Hand*, Baltimore, 1940, The Williams & Wilkins Company.
- Kanavel, Allen H.: *Infections of the Hand*, ed 6, Philadelphia, 1939, Lea & Febiger.
- Moseley, H. F.: *Shoulder Lesions*, ed. 2, Springfield, Ill., 1952, Charles C Thomas
- Moseley, H. F.: *Ruptures of the Rotator Cuff*, Publication No 106, American Lecture Series, Springfield, Ill., 1952, Charles C Thomas.
- Steindler, Arthur: *The Traumatic Deformities and Disabilities of the Upper Extremity*, Springfield, Ill., 1946, Charles C Thomas

Isolated Fractures

In avulsion fractures rest for 3 to 4 weeks in the relaxed muscle position is indicated.

Sacrum and Coccyx

Avoidance of pressure and bed rest for 2 to 3 weeks are required. Fractures of the coccyx are rare, but ligamentous injuries are common and are prone to be followed by a painful neuralgia, i.e., traumatic coccydynia. This is best treated by avoidance of pressure on sitting by use of an air ring supplemented by local diathermy. Excision of the coccyx may be necessary in chronic cases.

3. Sacroiliac pain.

4. Traumatic urethral stricture.

5. Coccydynia.

DISLOCATION OF THE HIP

Dislocations of the hip are not common and result from severe violence. The head of the femur may displace backward or forward out of the acetabulum or be driven centrally carrying the floor of the acetabulum with it.

Mechanism.—Posterior dislocations are produced when the patient is seated with the thighs flexed and adducted. The force is

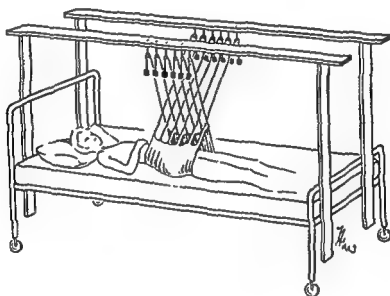


Fig. 386—Pelvic sling.

In all serious injuries of the pelvis, a firm canvas support should be provided when the patient becomes ambulatory. Ambulation on crutches is advisable before full weight-bearing is allowed. Any tendency to pain in the sacroiliac area merits in addition massage and diathermy treatments

Late Complications.—

1. Malunion with interference, in female patients, with the pelvic canal and parturition.

2. Traumatic arthritis of the hip.

transmitted up the femur from the knee, and the head is driven out of the acetabulum in an upward and backward direction. The head may be lodged upward on the dorsum ili or lower down into the sciatic notch. These accidents are seen in train, car and plane accidents, but may occur when heavy weights fall on the back of the patient who is buckled under by the force.

Anterior dislocations result from forced abduction of the thigh, the head going forward and upward onto the pubis or downward into the obturator foramen.

Associated Injuries.—**1. Visceral —**

- (a) Urethra.
- (b) Bladder. Intra- or extraperitoneal rupture.
- (c) Rectum.

2. Nervous — Injuries to lumbosacral plexus S 12. characterized by the clinical signs:

- (a) Toe drop.
- (b) Loss of Achilles' reflex

Clinical Picture.—The patient presents the general picture of shock in severe crushing injuries. Visible deformity is not often present. There may be pain localized to the pelvis on attempting to bear weight or the patient may localize the pain and tenderness to different parts of the pelvis when lateral or anteroposterior compression is applied.

The diagnosis is accurately made and any displacements of the fragments noted on x-ray examination, which is essential.

Because of the possibility of visceral injury, the patient is asked whether he has passed urine and if so whether blood stained. If urine has not been passed the patient is advised against doing so. The presence of blood at the urethral meatus or a perineal hematoma indicates urethral damage. Suprapubic tenderness or early abdominal rigidity suggests rupture of the bladder.

A rectal examination is necessary to ascertain injuries to this viscus, and by this route fractures of the ramus may be palpated.

Treatment.—

General—If it is necessary to move the patient, this is best performed after binding the pelvis to prevent displacement. Therapy directed toward shock may be required. The patient is placed on a firm mattress supported by fracture boards.

Visceral—Catheterization should be carefully carried out. Failure to enter the bladder indicates rupture of the urethra. If the catheter enters the bladder but fails to drain urine, an intraperitoneal rupture must

be suspected. The presence of a small quantity of blood-stained urine suggests extraperitoneal rupture and extravasation. Lacerations of the rectal wall found on rectal examination or vesical injuries require laparotomy and repair.

Fractures**PELVIC RING**

Fractures of the rami do not displace greatly and unite without residual disability after 3 to 4 weeks of bed rest.

Fractures of the anterior and posterior sections of the ring without displacement are best treated in bed for 4 to 6 weeks. A sling support often gives comfort to the patient.

Fractures of the anterior and posterior sections of the ring with displacement require treatment in a pelvic sling together with traction on the side of upward displacement of the innominate bone. This correction is best effected with skeletal traction through the lower end of the femur. Six to eight weeks are required for union in the reduced position.

In cases of anterior and posterior fractures from anteroposterior compression, a plaster spica applied in the lateral position (Watson-Jones) is an excellent method of reduction and fixation. Six to eight weeks are required.

Rim.**ACETABULUM**

Floor. In these cases the problem is how to avoid or minimize traumatic arthritis. When no displacement occurs, the treatment consists in gentle movements with avoidance of weight-bearing for 3 to 4 months. With central dislocation of the femoral head, reduction is carried out at the earliest moment by strong traction on the abducted leg. Sometimes open operative reduction is required.

Weight-bearing should be avoided for 4 to 6 months and the progress followed for several years to note the development of aseptic necrosis of the femoral head and traumatic arthritis.



Fig. 387B—X-ray of central dislocation.

Classification.—

1. Posterior.

- (a) High—onto the dorsum ilii.
- (b) Low—into the sciatic notch, below and behind the obturator internus.

2. Anterior.

- (a) High—onto the pubis.
- (b) Low—onto the obturator externus.

3. Central—carrying the floor of the acetabulum inward.

Clinical Picture.—

Posterior Dislocation—The lower limb in the high dislocation is $1\frac{1}{2}$ to 2 inches shortened, and is held adducted and internally rotated. The knee is therefore applied to the anterointernal aspect of the opposite

thigh just above the patella and the plantar flexed foot reaches the dorsum of the opposite foot. Attempts to abduct the limb are resisted by muscle spasm. When the head is displaced into the sciatic notch, the shortening is only $\frac{1}{2}$ to 1 inch.

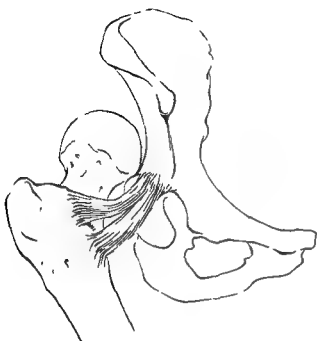
Anterior Dislocation.—The limb is $1\frac{1}{2}$ to 2 inches longer than normal in the obturator type and is held in abduction and external rotation. The pubic dislocation does not show such marked increase in limb length.

Central Dislocation.—The limb is held in slight flexion, and attempts at movements are resisted by muscle spasm.

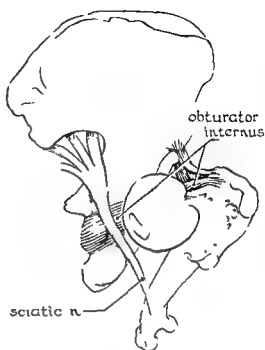
Diagnosis.—In most cases the history and clinical picture render diagnosis easy. X-ray examination is conclusive.



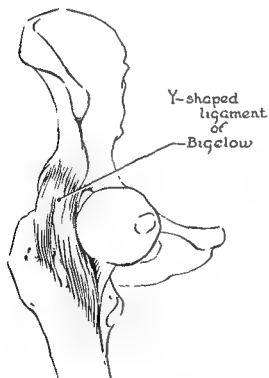
Fig. 388—Deformity of the posterior dislocation.



posterior dislocation
-gluteal position



posterior dislocation
below obturator internus



anterior dislocation
-pubic position



anterior dislocation
-obturator position

Fig. 387A—Type dislocations of the hip.

A.



B.

Fig. 390 —A. Congenital dislocation on right side. Shenton's line, which is shown in its normal arrangement on the left side, is broken on the right side.

B. The same case after reduction by traction. Note the increased slope of the acetabular roof and the delay in the development of the femoral head on the right side.

Treatment.—Bigelow demonstrated that the head dislocates anteriorly and posteriorly with the iliofemoral ligament as the pivot. This ligament rarely ruptures.

Reduction should be carried out with complete relaxation secured by appropriate anesthesia. The patient is placed on the floor and reduction is achieved by the use of long axis traction on the flexed hip and knee associated with circumduction and sometimes direct manual pressure on the displaced head.

Postoperative fixation in a plaster spica or in suspension with light traction for 3 to 4 weeks is desirable.

Avoidance of weight-bearing for 3 to 4 months will minimize the tendency to aseptic necrosis of the femoral head and traumatic arthritis.

Complications.—Fracture of the posterior acetabular rim frequently occurs with posterior dislocations. No special treatment is indicated unless the fragment is large and remains displaced after reduction of the dislocation. Operative fixation is required in these cases.

Aseptic necrosis of the femoral head may result from rupture of the ligamentum teres and vascular derangement. Traumatic arthritis invariably occurs. The progress should be followed by regular x-ray examination.

Sciatic nerve damage sometimes occurs by direct contusion or stretching in the posterior dislocations.

Myositis ossificans may develop in torn capsular and muscular tissues around the joint.

CONGENITAL DISLOCATION OF THE HIP

Congenital dislocation of the hip is the commonest congenital dislocation. It occurs more frequently in girls than boys in the ratio of four to one and one out of every three cases presents a bilateral lesion. There is a geographical and racial incidence noted. This congenital anomaly is especially com-

mon in northern Italy where Putti has made extensive studies and contributions on this subject. It is rare among Negroes. There appears to be a hereditary tendency to this defect.

The term congenital dislocation includes the various degrees of displacement of the head from its normal position and thus includes subluxations.

Pathological Anatomy.—The acetabulum is shallower, and its roof presents a greater obliquity, than normal. There is a postero-superior defect of the acetabular rim. In many cases the acetabulum is obliterated by fibrous tissue and capsular adhesions. There is a false joint above the acetabulum on the dorsum ilii.

The upper end of the femur is also abnormal. The epiphysis of the head, which appears in infants between six and eight months, is hypoplastic and sometimes undergoes an osteochondritic process. In the early stages before ambulation the head is more laterally placed from the floor of the acetabulum than normal. In the later stages

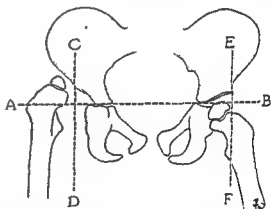


Fig. 389.—The method for localization of the head of the femur in relation to the acetabulum. AB = horizontal line through points of fusion in the acetabula. CD and EF = perpendicular lines drawn from the superior rims of the acetabular roofs. Normally, the head lies inferior to the horizontal line and medial to the vertical line. In congenital dislocation, as shown on the right side, the head lies superior to the horizontal line and lateral to the vertical line. The head, in such cases, is usually late in development.

After this time, the treatment consists of gentle reduction of the dislocated head by traction followed by positioning the limbs in full internal rotation until stability is secured which averages 9 months. This position is maintained by plaster fixation which is changed at 3 monthly intervals. As soon as the reduction is stable dynamic splinting in abduction may be employed instead of plaster fixation (Dennis Browne).

In those cases that relapse or where reduction was impossible, it may be achieved by the same method with the addition of cross traction at right angles to the femoral shaft. This group often shows a high degree of anteversion of the neck which may require correction by external rotation osteotomy.

When the patient presents later with such dislocations a variety of reconstructive procedures may be employed

THE FEMUR

The femur is the largest and strongest bone in the body and the muscles acting upon it are most powerful

Fractures may involve (a) the upper end, (b) the shaft, or (c) the lower end.

The Upper End of Femur

Separation of the upper femoral epiphysis occurs in the overweight adolescent aged 10 to 16 years.

Fractures of the upper end of the femur in relation to the hip joint are common accidents of old age. Formerly, and sometimes still called, the "unsolved fracture," this injury converts the ambulatory aged into bed patients with the usual complications of such confinement. The introduction of the Smith-Petersen nail for internal fixation has greatly diminished the morbidity and mortality of these fractures.

Classification.—

1. Separation of the upper femoral epiphysis

2. Subcapital.
3. Transcervical.
4. Intertrochanteric.

Mechanism.—

Upper Femoral Epiphysis (Adolescent Coxa Vara).—Separation or slipping of this epiphysis may occur acutely or gradually. The head rotates posteriorly and the femoral shaft and neck displace forward. An endocrine factor, in addition to obesity, has been advanced to explain the causation. The result, if uncorrected, gives rise to diminution in the angle of the neck and shaft, i.e., a coxa vara. This is to be differentiated clinically from osteochondritis of the femoral head (Perthes' disease) which occurs at an earlier age, i.e., 5 to 10 years.

Subcapital Fracture.—The thin old lady, past her biblical span of life, is the typical case to present the subcapital fracture. The predisposing factor is atrophy of the bony structure in the femoral neck. A sudden trip on the scatter rug, polished floor or bath, the bone gives way, and the patient sinks to the floor. There the doctor finds her, unable to rise because of pain and with the affected limb externally rotated in characteristic deformity.

Transcervical and Intertrochanteric Fractures may occur in the aged but are also seen in younger individuals. The mechanism of injury is frequently similar, but a direct fall on the trochanteric region or a forcible twist of the leg, concentrating the force in this area, may cause such fractures

The term *intracapsular* occurs in the literature for the subcapital type and *extracapsular* for the others. These terms had their importance during a period when the view was stressed that access of synovial fluid to the fracture line was a factor in causing nonunion. Also it was known that the subcapital fracture had the greater interference with circulation to the femoral head. Today it is realized that the speed of union diminishes as the fracture line is placed nearer the anatomical neck, largely because

the shape of the head is altered from pressure changes, being flattened on its medial and posterior aspects. The normal anteversion of the neck is increased and the neck points almost directly forward. In cases at 20 to 30 years of age the x-ray appearances of degenerative arthritis, characterized by cystic areas and osteophytic changes, are noted in the head.

The capsular changes have been studied at operation, autopsy and lately by arthrography. In some cases the capsule is drawn out in hourglass arrangement, and part of the capsule intervenes between the head and the false socket on the ilium. The hourglass constriction permits the passage of the ligamentum teres carrying part of the blood supply to the head. This constriction is a barrier to manipulative replacement as it intervenes between the head and acetabular socket on closed reduction.

It is generally agreed that before ambulation has occurred the head is usually capable of being replaced in the acetabulum by abduction of the limbs. With the passage of time, adaptive structural changes occur which make anatomic position more difficult to obtain and retain. Early diagnosis will depend on the careful examination of the hip joints of all infants with radiological studies, if deformity or limitation of movements is found. If the preluxation state is demonstrated, treatment can be initiated at the earliest moment.

Clinical Features.—Preambulant Period.—The radiological examination is the key to the discovery of the preluxation. At birth an increased obliquity of the acetabular roof can be noted. At one month any increased lateral displacement of the upper and inner corner of the femoral metaphysis may be seen and after 6 to 8 months the delay in the development of the upper femoral epiphysis or its smaller size can be demonstrated.

Ambulant Period.—As soon as the child begins to walk, a definite limp will develop. In the unilateral case there will be a lurch to the affected side. There is a *positive*

Trendelenburg test which means that when the child stands on the affected leg and raises the other from the ground the pelvis will lower on the normal side because of the lack of the stabilizing influence of the gluteal muscle group. Clinical examination will also disclose an increased number of folds on the medial and posterior aspects of the thigh. Palpation will reveal the absence of the head in Scarpa's triangle and the femoral pulsation will be more difficult to obtain for this reason. The head may be palpated in the gluteal area. Movements of the hip are altered and there is limitation of abduction and internal rotation with increased adduction and external rotation. It may be possible to telescope the upper end of the femur. There is a greater than normal prominence of the great trochanter, and measurements will reveal that the trochanter rides above Nelaton's line joining the anterior superior spine to the ischial tuberosity.

X-ray at this period will show that the head is more superiorly and laterally placed than normal and the line of Shenton is broken.

In the bilateral case the child walks with a waddle. There is a greatly increased lumbar lordosis and protuberant abdomen, giving the appearance that the trunk is falling forward on the posteriorly displaced hip mechanisms. There is a bilateral positive Trendelenburg test.

Diagnosis.—The work of Putti has shown that by public education this anomaly can be diagnosed in the early months of life if the possibility is suspected and radiographic studies made. After ambulation has begun the diagnosis is made on the characteristic limp and clinical features mentioned above.

Treatment.—If this congenital anomaly is diagnosed in the preluxation period, positioning the limbs in abduction and internal rotation on various types of mattresses, cushions or braces for periods of six months or more can effect a cure. This is only of value within the first six months of life.

of the femoral neck discloses tenderness. Manipulation of the limb gives bony crepitus and extreme pain except when impaction is present. X-ray examination gives the accurate location of the fracture site and displacements.



Fig 391B—X-ray of Legg-Perthes' disease.

Treatment.—

Separation of the Upper Femoral Epiphysis.—A period of observation in the orthopedic ward is indicated. If seen at the time of an acute displacement, manipulative reduction and introduction of a Smith-Petersen nail is recommended. Some prefer manipulative reduction and fixation in a plaster spica until synostosis of the epiphysis occurs. Careful x-ray follow-up is required in all cases.

In cases seen early where the separation is gradual, bed rest and avoidance of weight-bearing are essential. A great saving of time is achieved by insertion of a nail. When seen late with gross displacement, open operative correction followed by nailing is indicated.

Subcapital Fractures.—Such patients should be transferred to hospitals where facilities for operative therapy are available. The ideal treatment is the insertion of a Smith-Petersen nail at the earliest convenient moment after the general condition of the patient has been evaluated. Delay of surgical intervention often does more harm than good.

Transcervical and Intertrochanteric Fractures.—Transcervical fractures are sometimes impacted in abduction. These can be treated in a plaster spica for 3 to 4 months, union being assessed by x-rays. This conservative

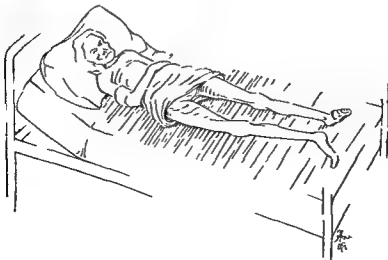


Fig 392.—Fractured neck of femur with typical deformity.

of difficulty in reduction and fixation as well as deficiency of circulation.

The subcapital fracture shows an eversion deformity of the limb of 45° because the capsule is still attached to the neck and this prevents full external rotation by the weight of the extremity. The transcervical and intertrochanteric fractures usually show the full external rotation deformity.

onset is not simultaneous. Examination shows the limb externally rotated, and there is limitation of internal rotation on passive manipulation.

Careful radiological examination in antero-posterior and lateral planes is required to diagnose the early stages. The late stages with marked displacement present little difficulty in diagnosis.

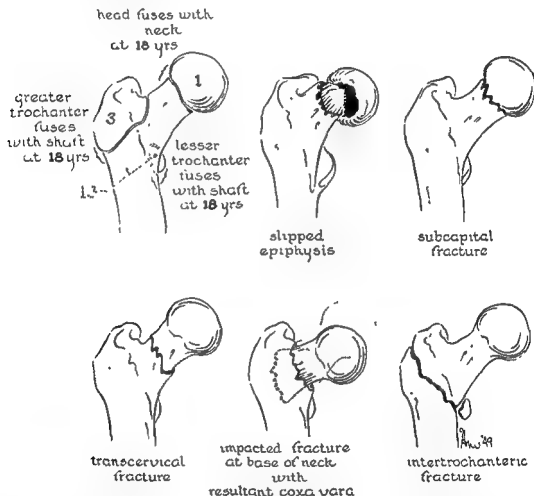


Fig 391A—Ossification, slipped epiphysis and fractures of the upper end of the femur.

Diagnosis.—

Separation of the Upper Femoral Epiphysis most commonly occurs in the Fröhlich type of obesity between the ages of 10 and 16 and should be suspected when such a patient complains of acute or chronic disability in the hip with consequent limp. There may or may not be a history of trauma, and the condition may be bilateral although the

Subcapital Fractures may be impacted or grossly displaced. The old lady with the everted limb and pain in the groin is typical. All such cases should be regarded as possible fractures until proved otherwise by careful x-ray examination.

Transcervical and Intertrochanteric Fractures present the full external rotation deformity with pain in the groin. Palpation

is satisfactory nonunion can result from failure to immobilize the fracture until union is complete. This was more frequently observed before the use of the Smith-Petersen nail. The use of traction or even the plaster spica did not hold the fragments in apposition. Since the introduction of the nail, with the use of impaction during the operation, adequate fixation is usually secured. Sometimes, however, the nail fails to secure sufficient purchase in the atrophic cancellous bone of the head, and a shearing force develops at the fracture line. This is especially true if the nail is horizontally placed, i.e., the fracture nailed in *varus* deformity.

Treatment of nonunion with a viable head is by introduction of a second nail above or below the first and replacement of the first by a tibial bone graft. Impaction of the fragments must be secured and immobilization is best effected by a plaster spica. Union must be carefully evaluated by x-rays.

Nonunion with a nonviable head is more difficult to treat. The aseptic necrosis is indicated by relatively increasing density of the head which may be noted after six months or longer. This can occur whether the fracture has been nailed or not and is due to interference with the circulation to the head. Union in such cases will not occur, and once the diagnosis is made a McMurray osteotomy or one of the many reconstructive procedures is the treatment of choice.

Complications of Nailing.—*Extrusion of the Nail.*—In cases where aseptic necrosis of the head develops or where a shearing force at the line of fracture occurs, the nail moves in the line of least resistance. In cases where the head of the nail is fixed to the shaft, the nail will penetrate into the joint. Where it is not so fixed, the nail will be extruded. This is due to the fact that the more vascular bone of the shaft area atrophies more rapidly around the nail and therefore permits the extrusion. Treatment depends upon the individual case and follows the principles outlined under nonunion.

Penetration of the Nail.—In cases where the nail has been hammered too far and has penetrated the joint, withdrawal is required because of the painful arthritis which develops.

Fracture of the Nail.—Occasionally the nail may break and require removal or replacement.

Shaft of the Femur

The shaft of the femur is the longest and strongest bony structure in the body. It has normally an anterior and lateral bowing. Fractures result from great violence and are difficult to reduce. Following reduction they are difficult to maintain in position because of the powerful muscle forces acting upon them. There is a tendency for fixation of the quadriceps to the healing area with consequent limitation of function. This is one of the factors causing the stiff knee which is the chief disability after such injuries.

Mechanism.—Direct violence of great severity is the usual cause. Indirect violence giving a torsion force and resulting spiral fracture accounts for a smaller number.

The displacements taken up initially depend on the direction of the causative forces, but the powerful muscles gradually pull the fragments into positions of characteristic deformity.

Fractures of the upper end present, flexion, abduction, and external rotation of the upper fragment and adduction of the shaft produced by the adductor magnus. Because of this, reduction and fixation are best arranged in flexion and wide abduction of the whole limb.

Fractures of the shaft proper in its middle half present adduction of the lower in relation to the upper fragment with overriding caused by the spasm of the hamstrings and quadriceps. External bowing is produced frequently in these cases. Traction must therefore be maintained in moderate abduction.

therapy may be indicated in the younger patient, 40 to 60 years, but here too internal fixation is probably the ideal method

Intertrochanteric fractures constitute a group in which the lines of fracture run in variable directions in relation to the greater and lesser trochanters. Reduction may be obtained by manipulation, skeletal traction, or by open operation. Fixation may be obtained by plaster spica, maintenance of traction with suspension of the limb, or by internal fixation. The problem of securing union is not so difficult here as in the subcapital and sometimes the transcervical fractures in older patients

mediately, or be lifted into a chair. This will obviate pressure sores from prolonged rest in one position.

Ambulation on crutches is determined by the patient's capacity. Often this is too dangerous to consider.

Union occurs in 3 months in the intertrochanteric, 4 months in the transcervical, and 4 to 6 months in the subcapital fractures. However, these are only arbitrary time factors, and union is best judged by radiological examination. Internal fixation is only a means of maintaining position. Time and the healing process are the essentials for union.



Fig 393 —X-ray of a displaced subcapital fracture before and after insertion of Smith-Petersen pin

The amount of metallic fixation in these cases is determined by the strength of the tongue of bone running up from the shaft. If this is strong, a nail alone will suffice. Usually, however, a side plate on the shaft is necessary as a supplement to give firm support to the nail in the neck and head fragment

Postoperative Therapy.—If the fracture is well nailed, patients with the above types are allowed to move freely in bed. Six to eight weeks of such confinement is conservative in this type of treatment. If necessary, the patient may be allowed to sit up in bed im-

mediately, or be lifted into a chair. This will obviate pressure sores from prolonged rest in one position.

Complications.—**Nonunion.**—Nonunion of the subcapital and transcervical fracture is the most serious complication. This may occur with a viable or nonviable head of the femur and the prognosis and treatment differ in the two types

Nonunion with a viable head is the result of inadequate reduction or inadequate fixation or both. Certain cases cannot be reduced by manipulation and require open exposure. Faulty x-ray technique may fail to indicate inadequate reduction. If reduction

If traction is employed, skeletal is preferable to skin traction. The Kirschner wire or Steinmann pin may be placed through the lower end of femur or upper end of tibia. Either site has its advantages and disadvantages.

spreading infection may cause inflammation and obliteration of this pouch with resulting stiffness of the knee.

It is technically easier to put the pin through the upper end of the tibia at the level of the tibial tubercle. When skeletal

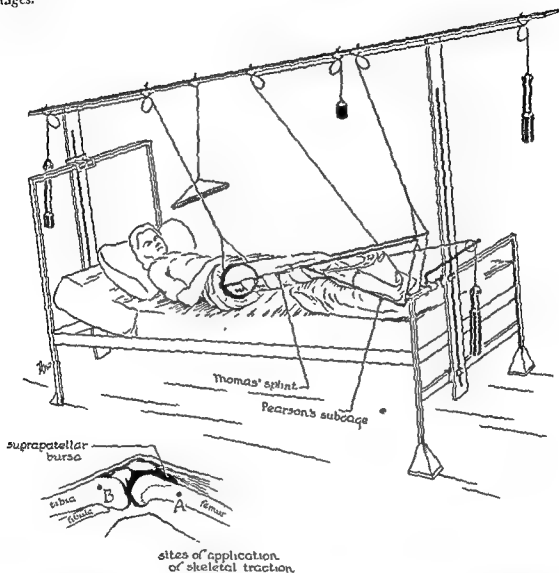


Fig. 395.—Balanced traction for fractured femur. Inset shows site for insertion of Kirschner wire or Steinmann pin, A, in femur; B, in tibia.

The pin should not be placed through the lower end of the femur in the vicinity of the fracture hematoma which may thus become infected.

The pin should be driven from the inner to the outer side to avoid injury to the femoral vessels in Hunter's canal and should not penetrate the suprapatellar pouch since

traction is applied at this site, however, the force is transmitted through the knee joint of which the ligaments may be stretched. This danger is avoided if the extension is not maintained too long or with too great force.

When the patient is under anesthesia the pin is introduced and traction is set up.

In supracondylar fractures, the lower fragment is angulated backward by the gastrocnemius. Reduction must therefore be carried out with these muscles relaxed by flexion of the knee and a counterforce applied to maintain the corrected position.

Clinical Picture.—The patient is in great pain and may be in shock with associated injuries to other structures. Deformity is sometimes marked, and the fracture area is surrounded by a large hematoma. Local tenderness and crepitus will be found. The patient is moved with great difficulty. X-rays reveal the type of fracture and displacements.

Definitive Treatment.—Various methods of treatment may be considered for shaft fractures, and the choice will depend on the judgment of the surgeon in charge.

1. Traction and countertraction.
2. Manipulation and plaster spica.
3. Open operation and internal fixation.
4. External-skeletal fixation.

Principles Underlying Therapy.—Reduction should be anatomical if possible in cases of femoral fractures, but this is sometimes impossible to achieve. Angulation must be avoided at all costs and the proper length of the limb maintained. Furthermore, alignment of the bones should be such as to avoid

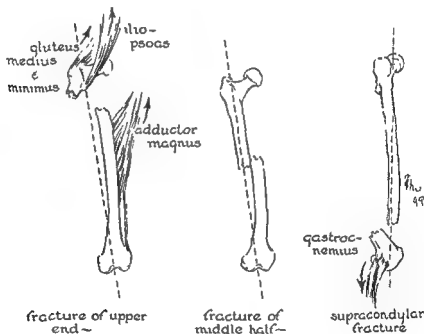


Fig. 394—Influence of muscle pull on fractures of the femur.

Treatment.—

Emergency Treatment.—The limb should be splinted before moving the patient. A Thomas splint with fixed traction or a plaster spica is the ideal method and is used if the patient is to be transported long distances. If such facilities are not available, fixation of the two limbs together and both fixed to a rigid support can be used. Morphine should be given to alleviate pain.

abnormal stresses and strains on the adjacent joints above and below.

If reduction can be secured by conservative measures such as skin or skeletal traction, these methods should be employed. Sometimes interposed tissue or injuries to other bones or the patient's general state may demand the use of open operative reduction and internal fixation to facilitate aftercare.

traction through the knee or placement of the pin or wire too near the suprapatellar pouch. This stiffness may be minimized by maintenance of activity of all muscles of the limb during therapy.

When seen as a late complication, a trial of quadriceps drill and active movements of the knee is recommended before manipulation under anesthesia or quadriceps lengthening is considered.

THE KNEE

The knee is the joint of the lower limb most subject to injury in the course of athletic and occupational activities. Lesions of the soft parts greatly outnumber those of the component bones. As stability in various positions of the knee constitutes the essential function of this joint, the power and co-ordination of the musculature are the secrets of success in the treatment of injuries to its mechanism.

Classification.—

- (a) Injuries of the Extensor Apparatus.
 - Hematoma of thigh.
 - Rupture of quadriceps
 - Patellar lesions.
 - Rupture of patellar ligament.
 - Osgood-Schlatter's disease.
- (b) Injuries of Other Soft Tissues.
 - Synovitis.
 - Hemarthrosis.
 - Internal lateral ligament.
 - Cruciate ligaments.
 - Semilunar cartilages.
- (c) Fractures of Component Bones
 - (1) Femur—Lower femoral epiphysis.
 - Condylar and intercondylar; articular; osteochondritis.
 - (2) Tibia—Bumper fracture.
 - Lateral tuberosity.
 - Tibial spine.
 - (3) Patella—Stellate; transverse; chondromalacia.
- (d) Dislocations.
 - Knee.
 - Patella.

Injuries of the Extensor Apparatus

HEMATOMA OF THE THIGH

Large volumes of extravasated blood may accumulate in the quadriceps mass as a result of a direct blow or kick at football; 500 to 1,000 c.c. is not unusual.

Diagnosis.—The history of the injury together with a swollen tender thigh is typical. Fluctuation is present in the early cases.

Treatment.—If seen within 2 to 3 days aspiration with a wide bore needle or evacuation through a small incision is indicated. A compression bandage is applied for 12 to 24 hours followed by quadriceps drill. Bed rest for 2 to 3 days and then ambulation on crutches and graduation to normal activity is the postoperative routine.

If seen later, the hematoma may be clotted, and it is best to apply hot packs and initiate quadriceps drill. The hematoma begins to liquefy between 10 to 14 days and the serum may then be evacuated. If the femoral periosteum has been raised, myositis ossificans sometimes develops. The use of a Thomas caliper to prevent knee movements is then beneficial, and the progress must be followed by x-rays at regular intervals.

RUPTURE OF THE QUADRICEPS

Rupture of the quadriceps from the patella occurs in the older patient from a sudden muscular contraction. The separation may be of the central part alone, involving the rectus femoris and crureus, or the lateral and medial expansions may separate as well. The condition may be bilateral. Degenerative changes at the patellar insertion constitute the predisposing cause.

Diagnosis.—The diagnosis is made on the history of injury together with a hemarthrosis and fluid hematoma at the upper pole of the patella. The patient is able to flex the knee but cannot lift the extended leg. The area of separation can be felt as a deep groove above the patella.

The fracture is manipulated to the correct length of limb and correct position, and traction and countertraction are instituted. Approximately one-seventh the body weight is adequate for purposes of extension, but x-ray follow-up gives the best indication of the amount to use in the individual case.

It is wise to avoid pin traction from one site of longer duration than 4 to 6 weeks.

When x-rays show satisfactory position and good callus formation (8 to 12 weeks), traction is replaced by a plaster spica or Thomas caliper. The Thomas caliper may be used subsequently as a support when the patient becomes ambulatory on crutches. In the young child below the age of 4 years, suspension traction gives the best results

permitted until 6 to 8 weeks have passed and protective splinting is necessary for approximately 4 to 6 months.

The subtrochanteric fracture presents a special problem and is probably best treated by open operation and internal fixation. Intramedullary nailing may be considered for this type of case.

Some cases in the young show spiral fractures with little displacement. These can be treated by plaster spica fixation.

External skeletal fixation may also find a place in selected cases.

It is most important in fractures of the femur to maintain the activity of the quadriceps muscle and at the earliest moment obtain the return of knee function.

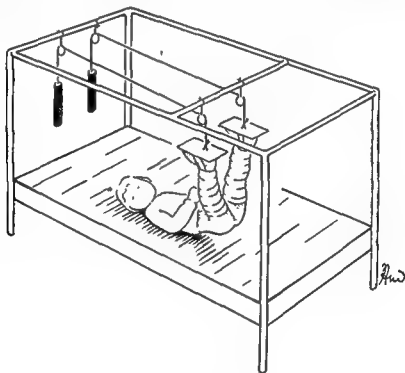


Fig 396—Bryant's suspension

When it is decided that open operation is indicated, the anterolateral approach is employed and a 1/2 to 3/4 inch screw plate is applied with the screws transfixing both cortices.

A Thomas caliper may be used as a splint in the postoperative period preferably to a plaster spica. Ambulation should not be

Transverse fractures of the femur usually unite more slowly than the spiral fractures. In the adult, the time loss from work averages 8 to 12 months.

Complication.—The chief complication is stiffness of the knee which should be prevented by avoiding prolonged or excessive

tubular cast with the knee in a position of extension. The cast should be maintained for 4 to 6 weeks, and this is followed by re-education of knee movements.

small fragment at the upper or lower pole is best excised.

When the patella is comminuted or where a smooth articular surface cannot be ob-

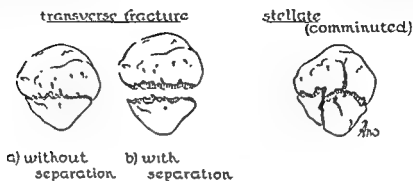


Fig. 398.—Type fractures of the patella.

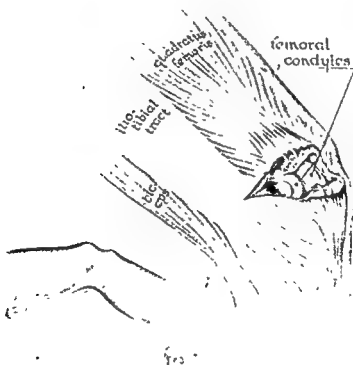


Fig. 399—Transverse fracture of patella with rupture of lateral expansions permitting separation

Transverse fractures with separation are best treated by open operation. The hemarthrosis is evacuated and the patella and lateral expansions meticulously sutured. The patella must be so approximated as to give a perfect articular surface. Sometimes a

tained, excision of the patella is indicated. The extensor tendons must be plicated and the continuity with the patellar tendon carefully obtained.

Postoperative therapy consists of a plaster cast with the knee completely extended

X-ray examination may show an abnormal tilting of the patella with the upper pole separated away from the femoral condyles.

Treatment.—Treatment consists of early exploration with evacuation of the hemarthrosis and careful suture of the avulsed quadriceps using drill holes in the patella. Fascial repair is sometimes indicated especially in the late cases where the diagnosis has been overlooked.

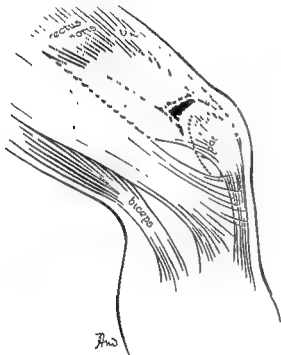


FIG 397.—Rupture of the quadriceps femoris insertion

PATELLA

The patella is the largest sesamoid bone in the body and constitutes a fulcrum over which the quadriceps expansion passes to the patellar ligament and tibial tubercle. It presents a bony barrier to injuries directed from the anterior aspect and determines the normal cosmetic appearance of this region.

FRACTURES

Mechanism.—Fractures of this bone may result from a direct blow or fall whereupon it is crushed against the intercondylar area of the femur. Such forces result in a stellate

fracture but it must be stressed that damage to the articular cartilage of the patella and intercondylar surface of the femur may be the most important component of the injury.

Sudden contraction of the quadriceps with a flexed knee may break the patella transversely with minimal or maximal separation of the fragments. The extent of bony separation indicates the degree of rupture of the lateral expansions of the quadriceps. The shape of the patella appears to predispose to knee injury, and some patients are subject to bilateral and even recurrent fractures.

Clinical Picture.—The region of the knee is grossly swollen, and evidence of bruising may be present. The injury may be associated with a wound compounding the fracture or communicating with the prepatellar bursa. Hemarthrosis is usually present. In transverse fractures with separation a groove is palpable between the fragments which increases when the patient flexes the knee or attempts quadriceps contraction.

The patient usually is unable to lift the extended limb.

Diagnosis.—The diagnosis is readily made on the clinical picture. X-ray examination in different planes affords conclusive proof and indicates the degree of comminution and separation.

Treatment.—The perfect result to be obtained from the treatment of patellar fractures is determined by three factors.

- 1 Restoration of the continuity of the extensor apparatus

- 2 Preservation of the fulcrum of the patella which gives the power in the final 5 to 10° extension.

- 3 Reformation of a smooth gliding articular surface between patella and femur.

In stellate or transverse fractures without displacement, this result may be obtained by the use of hot packs, compression bandages, and bed rest for 2 to 3 days, followed by quadriceps drill and early ambulation in a

Dislocation may occur as a single accident from sudden muscular contraction or the condition may be recurrent. In the recurrent cases the lesion may be bilateral. Medial dislocation can occur in cases presenting genu varum.

Mechanism.—In most cases the resultant force of the quadriceps contraction tends to displace the patella to the lateral side. In the complete displacement the patella rotates 90° or more around the lateral condyle.

Diagnosis.—When the patella is dislocated, the knee is held locked in a slightly flexed position. The abnormal position of the patella is seen. Any genu valgum deformity is noted and x-rays are taken to show the characteristics of the patella and femoral condyle.

Treatment.—Acute dislocation is readily reduced by direct pressure in a medial direction. The patient should then be placed on quadriceps drill to develop the musculature. This should also be initiated in recurrent cases and a firm bandage applied for a period if the patella is unstable.

In most recurrent dislocations operation is eventually required. This consists of transposition of the patellar tendon with its tibial attachment into a slot cut more medially on the tibia. The capsule at the same time is plicated on the medial side. This places the line of quadriceps pull more directly in line with its tibial insertion.

RUPTURE OF THE PATELLAR LIGAMENT

This can occur at the patellar or tibial attachment with or without a fragment of bone. The diagnosis is usually obvious. Treatment consists of careful suture followed by splinting of the knee in extension until firm union has occurred (6 to 8 weeks).

OSGOOD-SCHLATTER'S DISEASE

This is a sprain of the patellar tendon attachment to the tibial tubercle. The tubercle develops its ossification as a separate center or as a tongue-shaped extension of the

upper tibial epiphysis. This epiphysis fuses at 17 to 18 years. Before this period and especially from 12 to 15 years of age, athletic youths may develop pain and swelling of this area associated with limitation of knee movements and consequent limp.



Fig. 401.—Photograph and x-ray of Osgood-Schlatter's sprain

Clinical examination shows a prominence of the tibial tubercle more marked than on the opposite side. The swelling may be red-dened and tender. All grades of severity are seen.

for 4 to 6 weeks with quadriceps drill enforced. After splintage is discarded, re-education of knee movements is initiated.

CHONDROMALACIA OF THE PATELLA

Chondromalacia of the patella is a clinical entity receiving more and more attention as a cause of internal derangement of the knee. As a result of bruising of its articular surface by a fall or blow on the kneecap, the cartilage degenerates and sequesters giving rise to loose body formation. With separation of cartilaginous fragments, recurrent effusions develop and traumatic arthritis ensues.

Diagnosis.—The study of x-ray films carefully taken to show the articular surface in various planes may disclose irregularities which, with the clinical picture, concludes the diagnosis. The presence of loose bodies in the joint is suggestive.

Treatment.—Exploration of the knee joint is indicated and all components of the joint must be carefully studied through a medial parapatellar incision.

If the articular surface is not grossly diseased, the cartilage is shaved off down to compact bone to give a smooth surface. In some cases, however, the damage is so severe that excision of the patella must be carried out.

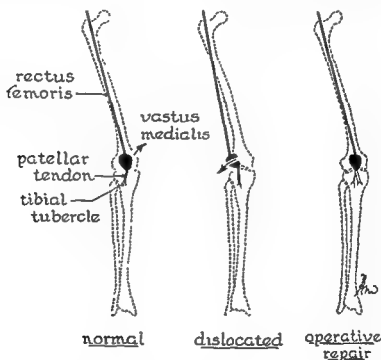


Fig. 400—Mechanism of recurrent dislocation of patella

Clinical Picture.—The patient may be an active youth with symptoms and signs of internal derangement. There may be complaint of pain centrally located in the knee deep to the patella. This is accentuated by movements of the knee with the patella compressed against the femur. Crepitus may be elicited in this maneuver.

An older patient may present with localized patellofemoral arthritis.

In all cases, a careful search for loose fragments of cartilage is made and their removal from the joint must be complete.

DISLOCATION OF THE PATELLA

Dislocation of the patella is usually to the lateral side and is predisposed to occur in patients with valgus deformity or where the patella is small, highly placed, and the lateral femoral condylar bu

ment of this ligament and separates partially or completely in varying degrees of valgus strain of the joint. This constitutes the first stage, which passes on to external condylar or tibial fractures or lateral dislocations of the knee.

The causative force may be a tackle at football from the side or a bumper injury from a car.

Diagnosis is made on the type of injury and the location of the tender point on the femoral condyle. In mild cases this may constitute the clinical picture. In severe cases the ligament is completely separated. Lateral instability is present. A serous effusion or hemarthrosis indicates the serious nature of the injury. The knee is held flexed and the patient bears weight with pain and a limp.

Elevation of the inner side of the heel 3/16" and in-toeing will relieve the strain when weight-bearing begins.

CHRONIC POSTURAL STRAIN AND STIEDA-PELLEGRINI'S SYNDROME

Both these clinical entities present the same clinical picture. They both occur in valgus strain of the knee of chronic type, whether in women of the sthenic type from the relative genu valgum or from the transmitted strain of pes valgus.

The patient complains of pain in the knee with inability to flex or extend the joint completely. There is the consequent limp, increasing with overuse. Tenderness is present over the femoral attachment and along the ligament.

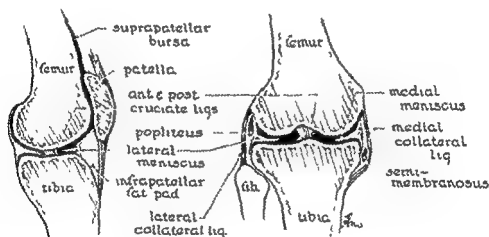


Fig 403—Sagittal and coronal sections of knee.

Treatment.—Mild cases require little more than a firm support. Complete ruptures with separation are best sutured by open operation followed by plaster immobilization for 4 to 6 weeks and concomitant quadriceps drill.

The majority of cases present an intermediate group. No instability is present but the final 20° of extension and complete flexion are painful. Treatment consists of heat to the affected area, quadriceps drill, and firm bandaging. Ambulation on crutches may be helpful for a week or more.

X-rays may show degenerative arthritic changes. In the latter syndrome, an area of calcification will be seen at the femoral attachment.

Treatment consists in diminishing the valgus strain. This may be accomplished by general measures such as the reduction of excessive weight. Raising the inner side of the heel and sole of shoe 3/16" and teaching the patient to in-toe will diminish the aggravation. With this is associated re-education of the muscular balance to both lower limbs working from the foot musculature.

X-ray examination may be negative or show a fragmentation of the tibial tubercle which with time undergoes the creeping substitution seen in the osteochondritic process.

Treatment consists of rest to the part. In mild cases strapping suffices. In severe cases with separation of the epiphysis, reddening and swelling, a tubular cast with the knee in extension is applied for 6 to 8 weeks.

If the severe cases are not so treated, a permanent failure of fusion of the bony tubercle may result. This is marked in later years by recurrent pain on excessive use and limitation of functional capacity. This results from the sprain of the central attachment of the quadriceps tendon, the lateral expansion attachments to the tibia being undisturbed.

X-ray reveals the separated tubercle and treatment consists of roughening its deep aspect and fusing this to the tibia. Excision of the fragment with suture of the central tendon to a raw bed on the tibia is an alternative procedure.

Injuries of Other Soft Tissues

SYNOVITIS

A serous effusion may occur in the knee as an acute or recurrent condition. The acute process may result from any irritation to the synovial membrane resulting from a sprain or from the presence of some mechanical derangement in the joint. Loose bodies or torn semilunar cartilages may be the irritant factors and an intra-articular lesion should always be suspected in the recurrent cases.

Treatment.—Aspiration of the fluid, followed by the application of a firm elastic support and quadriceps drill, is the treatment of choice. Recurrent effusions may require exploration of the joint to ascertain the cause.

HEMARTHROSIS

The knee may fill with blood as a result of a severe sprain, rupture of cruciate ligaments or fracture communicating with the

joint. A tense joint may develop rapidly within one hour, whereas serous effusions take the greater part of a day.

Clinically, the joint is held in the position of maximum joint capacity, i.e., 160°. There are diffuse tenderness and considerable pain on attempted movement. X-rays should be taken to exclude bony injury.

Treatment.—Aspiration should be performed. When the joint is lax, further examination may be carried out to ascertain the extent of injury. A firm bandage and quadriceps drill is the routine postoperative therapy. Exploration may be advisable.

INTERNAL LATERAL LIGAMENT

This ligament is subject to acute partial and complete tears. Many cases of prolonged disability of the knee are due to chronic postural strain of its femoral attachment. Calcified deposits may occur at this point and give rise to a syndrome named after Stieda and Pellegrini.

Mechanism of Rupture.—The femoral is much weaker than the broad tibial attach-

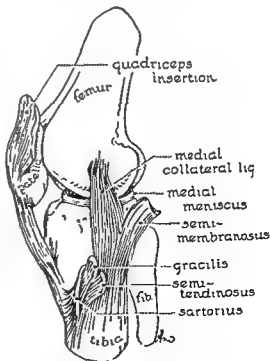


Fig 402—Medial aspect of knee.

game. He felt a sickening pain at the time and possibly heard or felt a click in his knee. The knee locked in a position of flexion and he was unable to carry on. When seen shortly afterward the patient is in pain. The knee is held by muscle spasm at 140° to 150° and attempts to walk on it or straighten it cause severe pain. Palpation over the inner joint line discloses a point of maximum tenderness.

Diagnosis is made on the typical clinical picture with the locked knee and tenderness localized to the joint line.

described by McMurray is of great diagnostic significance in doubtful cases. There may be a history of recurrent locking, recurrent effusion or repeated giving way of the joint.

Pneumoarthrograms may be helpful in cases of doubt.

Differential Diagnosis.—

1. *Quadriceps Insufficiency*.—After all injuries of the knee, there is rapid atrophy of the quadriceps unless this is prevented by muscular drill. Insufficiency of the extensor

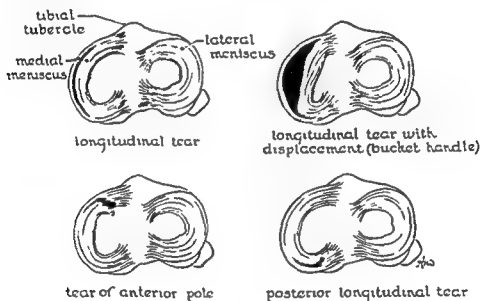


Fig. 405.—Common types of torn menisci.

Manipulation of the knee through a full range of movement under anesthesia unlocks the joint and adds confirmation to the diagnosis. X-rays in these cases disclose no bony injury but are taken routinely to exclude articular fractures or loose bodies in the joint.

The case may be seen later, when the cartilage has been reduced. Here the diagnosis may be more difficult.

Characteristic findings are atrophy of the vastus internus associated with effusions in the joint and joint line tenderness.

Manipulation of the knee in flexion and extension with varying degrees of rotation may elicit a click to the fingers placed along the joint line on the affected side. This test

mechanism can result in recurrent effusion, giving way of the knee, and all the points in the clinical picture of internal derangement. When suspected as the cause, the therapeutic trial of quadriceps drill is prescribed as a diagnostic and curative measure.

2. *Sprain of the Internal Lateral Ligament* is the most important injury to differentiate in acute cases from injuries to the internal meniscus. The mechanism of injury here is a direct abduction or valgus strain whereas the injury of the cartilage is caused by a rotation strain. Confusion arises where the two are associated.

The point of maximum tenderness is over the femoral attachment in the ligamentous injuries, and procaine infiltration may be

upward. Local heat is an adjuvant as are active movements of the knee on bicycle or sliding seat

CRUCIATE LIGAMENTS

The cruciate ligaments are ruptured in marked displacements of the tibia on the femur. They are therefore associated lesions in the various dislocations.

The *anterior cruciate* is tight in extension and limits hyperextension. It is injured in forcible hyperextension of the joint.

The *posterior cruciate* is tight in flexion and is injured in the dashboard type of injury when the flexed tibia is driven backward on the femur.

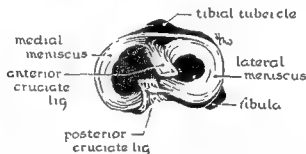


Fig. 404.—Attachment of menisci and cruciate ligaments to tibia

Clinical Picture.—The injury is usually a severe one with hemarthrosis present. Rupture of the cruciates results in an increased anteroposterior glide of the tibia on the femur in the flexed position.

Rupture of the anterior alone permits increased forward movement, rupture of the posterior alone increases posterior movement.

Treatment is determined by the concomitant injuries. Immobilization of the joint in a tubular plaster for three months associated with routine quadriceps drill is the most effective initial treatment. If associated with a dislocation of the knee, open operative suture of the ruptured soft tissue structures is indicated.

Late cases must be evaluated carefully and a thorough trial made of the stabilizing influence of quadriceps re-education before operation is considered.

Semilunar Cartilages

Injuries of the menisci constitute one of the most frequent and interesting groups of internal derangements.

Development.—The semilunar cartilages originate as a plate of mesoderm, which divides the joint into superior and inferior compartments. Later this plate is divided centrally by the ligamentum mucosum into a lateral and medial disc. The medial disc differentiates more rapidly and completely than the lateral disc into the semilunar-shaped structures, which serve to deepen the sockets for the femoral condyles. Arrest in differentiation is more common in the lat-

eral meniscus, which may present various forms to give the congenital discoid cartilage.

Mechanism of Injury.—The internal cartilage is injured 7 to 8 times more frequently than the lateral cartilage. This is because there is more tendency to an external rotation strain of the tibia on the femur in the flexed position of the knee. During this movement the internal cartilage is drawn between the articulating surfaces and, if ground between these surfaces with a shearing force, is fractured. Various types of meniscus injuries are produced as illustrated.

The external cartilage is injured by an internal rotation strain with adduction of the tibia on the femur.

Clinical Picture.—The patient is typically a young athlete, who suffered a severe rotation strain of the knee during a football

tures, there is a danger of injury to the popliteal vessels and nerves by the prominent shaft fragment. These epiphyseal injuries are uncommon but must be considered serious because of their tendency to interfere with growth and thus shorten the limb.

Reduction.—Manipulation in the flexed position of the knee with traction on the leg and countertraction to the thigh and direct pressure backward on the displaced epiphysis secures an easy replacement

and the tendency to restriction of knee movements as a complication.

Mechanism.—Falls from a height, a direct blow, or valgus or varus strain, give the various types of fracture. Shearing off of one condyle is most commonly produced by forceful displacement of the leg to the corresponding side associated with a compression force through the knee by the body weight.

Clinical Picture.—The knee presents a grossly swollen appearance with marked



Fig 407.—Osteochondritis dissecans.

The limb is splinted in a plaster cast with the knee at a right angle for four weeks. The cast is then changed to one with the knee in slight flexion for a further 3 to 4 weeks. Avoidance of weight-bearing for three months by ambulation on crutches tends to lessen the tendency to premature synostosis.

CONDYLAR AND INTERCONDYLAR FRACTURES

These fractures are serious because of their involvement of this weight-bearing joint

hemarthrosis. X-rays disclose the site of fracture and the displacements.

Treatment.—Aspiration of the hemarthrosis is beneficial. Reduction must be anatomical. If this is possible through tibial skeletal traction and external compression, conservative treatment is indicated. In the grossly displaced fractures, open operation with evacuation of extravasated blood and internal fixation by screw or bolt, securing meticulous alignment of joint surfaces, is the ideal treatment.

used to abolish the local pain and demonstrate the absence of mechanical block to knee movements. In cartilage injuries the tenderness is directly over the joint line and the McMurray test will be helpful in differentiation.



A.



B

Fig 406—Cyst of external cartilage A. Clinical photograph B Specimen

3. *Osteochondritis of the Femoral Condyle and Chondromalacia of the Patella* must be considered. X-ray examination disclosing the defect of articular cartilage and the presence of loose bodies in the joint demonstrate the chief differentiating points.

4. *Hypertrophy of the Infrapatellar Pad of Fat* may cause the symptoms of internal derangement when villous processes are nipped in the joint. Arthrotomy often is required to establish the diagnosis.

5 *Loose Bodies*.—Fibrous, cartilaginous and osseous loose bodies can give all the signs of internal derangement.

6 *Cysts of the Semilunar Cartilages*.—Ganglionic degeneration which chiefly occurs in the lateral meniscus will be indicated by the tense local swelling on the joint line

Treatment.—The locked knee caused by the displaced cartilage should be manipulated under anesthesia and the cartilage replaced. The joint is put through its full range of flexion and extension in varying degrees of rotation. This usually results in replacement of the displaced fragment.

Following manipulation a firm bandage is applied and quadriceps drill is initiated

In recurrent cases and in cases of initial injury where the diagnosis is certain, arthrotomy with complete removal of the cartilage is the ideal therapy. Postoperative treatment consists of immediate quadriceps drill. Ambulation on crutches may begin as soon as the patient feels able. Stitches are removed on the 7th to 8th day and flexion exercises begin between the 8th to 10th day. Good function is present after 3 to 6 weeks

Fractures of Component Bones

FEMUR

LOWER FEMORAL EPIPHYSIS

Separation of the lower femoral epiphysis occurs before its fusion (18 to 20 years). The mechanism of injury is a forcible hyperextension by which the epiphysis displaces forward with the leg, the lower end of the femoral shaft projecting backward into the popliteal space. As in supracondylar frac-

ARTICULAR FRACTURES (OSTEOCHONDritis DISSECANs, PAGET'S QUIET NECROSIS)

This is a specific entity causing internal derangement of the knee. It is one of the group designated osteochondritis and in the knee most commonly occurs on the anterolateral aspect of the medial femoral condyle. Most authorities regard the etiology as traumatic. An acute articular fracture may be produced possibly by pressure of the cruciate ligament in a severe rotatory strain. Possibly a contusion through the patella may be the mechanism. Following such an injury an area of cartilage and underlying bone begins to sequestrate and the patient may be seen either before or after the separation of the fragment. When first separated, there is cartilage on the one side and bone on the other. As time passes, fibrocartilage forms over the whole surface giving a uniform appearance to the loose body when seen at operation.

Clinical Picture.—The patient is a young male patient of athletic habits, who gives a history of injury to his knee some months previously. He now complains of the typical signs of internal derangement, i.e., recurrent effusion, weakness, locking or giving way.

X-ray examination is conclusive. A loose body is seen when separation is complete. In earlier cases the lateral view shows the sequestering fragment on the medial condyle.

Treatment.—Exploration is indicated. If the fragment is loose, it is removed and the joint thoroughly searched for other loose bodies. The condylar area is examined. Usually no surgical treatment is required to the healing area. If the fragment is in the process of separating, it should be removed and the area smoothed with gouge and chisel.

TIBIA

BUMPER FRACTURE

Bumper fracture is so named because of the frequency with which it is caused by the

car bumper in street accidents. When the leg is struck from the anterior aspect, the tibia is fractured just below the articular margin and often a longitudinal fracture makes communication with the joint.

When the bumper strikes from the lateral or anterolateral aspect a valgus strain of the knee develops and the tibial tuberosity on the outer side is depressed and sheared off. The varying stages of injury are shown in the accompanying diagram.

Clinical Picture.—This is similar to that presented by femoral condylar fractures. A hemarthrosis is usually present.

Treatment.—In cases without displacement of the tibial articular surface, aspiration of the hemarthrosis, manipulative reduction by traction in varus position and compression of the tuberosities when spread, followed by plaster immobilization in this position are used. Six weeks' immobilization is an average time and this is followed by re-education of knee function.

Where the lateral tuberosity is markedly displaced, open operation by a lateral parapatellar incision is performed. The displaced lateral meniscus is removed and the articular surface realigned. The limb is immobilized in varus position in full extension for six weeks. Re-education of knee movements follows. Weight-bearing in the displaced articular fractures is best avoided for 4 to 6 months to minimize the traumatic arthritis.

TIBIAL SPINE

Avulsion of the tibial spine occurs with anterior cruciate injuries. The fragment is displaced laterally and wedges between the lateral condyle and tibia, preventing complete extension.

The diagnosis is made by x-ray examination which demonstrates the displaced fragment.

Treatment.—Manipulation of the knee into complete extension and immobilization in a plaster cast for 11 to 8 weeks in this position may suffice. Open operation with

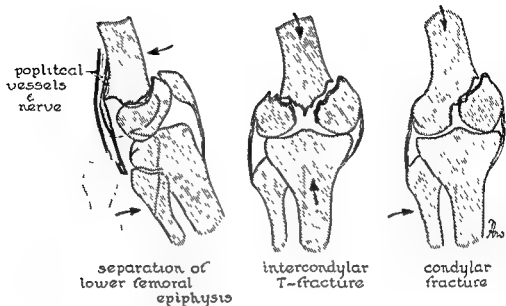


Fig 408 —Fractures lower end of femur

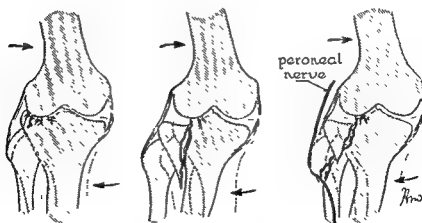
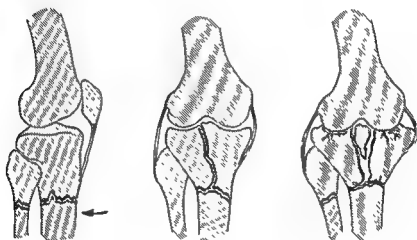


Fig 409 —Fractures upper end of tibia.

ARTICULAR FRACTURES (OSTEOCHONDritis DISSEANS, PAGET'S QUIET NECROSIS)

This is a specific entity causing internal derangement of the knee. It is one of the group designated osteochondritis and in the knee most commonly occurs on the anterolateral aspect of the medial femoral condyle. Most authorities regard the etiology as traumatic. An acute articular fracture may be produced possibly by pressure of the cruciate ligament in a severe rotatory strain. Possibly a contusion through the patella may be the mechanism. Following such an injury an area of cartilage and underlying bone begins to sequestrate and the patient may be seen either before or after the separation of the fragment. When first separated, there is cartilage on the one side and bone on the other. As time passes, fibrocartilage forms over the whole surface giving a uniform appearance to the loose body when seen at operation.

Clinical Picture.—The patient is a young male patient of athletic habits, who gives a history of injury to his knee some months previously. He now complains of the typical signs of internal derangement, i.e., recurrent effusion, weakness, locking or giving way.

X-ray examination is conclusive. A loose body is seen when separation is complete. In earlier cases the lateral view shows the sequestering fragment on the medial condyle.

Treatment.—Exploration is indicated. If the fragment is loose, it is removed and the joint thoroughly searched for other loose bodies. The condylar area is examined. Usually no surgical treatment is required to the healing area. If the fragment is in the process of separating, it should be removed and the area smoothed with gouge and chisel.

TIBIA

BUMPER FRACTURE

Bumper fracture is so named because of the frequency with which it is caused by the

car bumper in street accidents. When the leg is struck from the anterior aspect, the tibia is fractured just below the articular margin and often a longitudinal fracture makes communication with the joint.

When the bumper strikes from the lateral or anterolateral aspect a valgus strain of the knee develops and the tibial tuberosity on the outer side is depressed and sheared off. The varying stages of injury are shown in the accompanying diagram.

Clinical Picture.—This is similar to that presented by femoral condylar fractures. A hemarthrosis is usually present.

Treatment.—In cases without displacement of the tibial articular surface, aspiration of the hemarthrosis, manipulative reduction by traction in varus position and compression of the tuberosities when spread, followed by plaster immobilization in this position are used. Six weeks' immobilization is an average time and this is followed by re-education of knee function.

Where the lateral tuberosity is markedly displaced, open operation by a lateral parapatellar incision is performed. The displaced lateral meniscus is removed and the articular surface realigned. The limb is immobilized in varus position in full extension for six weeks. Re-education of knee movements follows. Weight-bearing in the displaced articular fractures is best avoided for 4 to 6 months to minimize the traumatic arthritis.

TIBIAL SPINE

Avulsion of the tibial spine occurs with anterior cruciate injuries. The fragment is displaced laterally and wedges between the lateral condyle and tibia, preventing complete extension.

The diagnosis is made by x-ray examination which demonstrates the displaced fragment.

Treatment.—Manipulation of the knee into complete extension and immobilization in a plaster cast for 6 to 12 weeks in this position may suffice. Open operation with

replacement of the fragment and suture may be preferred. The bony fragment may be excised.

DISLOCATIONS OF THE KNEE

The knee is not frequently dislocated. The causative force may be direct to the upper end of the tibia or indirect through the leg. Lateral dislocation is the most common, although anterior and posterior displacements of the tibia on femur occur.

Severe capsular and ligamentous damage results. The cruciates are disrupted.

The serious nature is due to frequent pressure on the popliteal vessels leading to gangrene. This occurs from the damage of the initial injury and sometimes is due to delayed reduction.

Clinical Picture.—The severe deformity is easily recognized. Careful examination to ascertain vascular and nervous injury is essential. X-rays are important.

Treatment.—The earliest possible reduction is imperative and this is readily obtained under general or spinal anesthesia. If anesthesia is not available, reduction should be attempted as a first-aid measure, using gentle continuous traction with the knee at 90°, associated with pressure on the displaced tibia. Open operative suture of the torn capsule and ligaments is being used more frequently on these cases.

Postoperative therapy consists in protection of the capsular tissues from strain until well healed (three months), associated with quadriceps drill.

TIBIA AND FIBULA

Fractures of the tibia and fibula are of frequent occurrence. The subcutaneous position of the tibia accounts for the fact that its fractures are more often open or compound in nature than those of any other major bone.

Mechanism.—Fracture of either bone singly is usually the result of direct trauma. Both bones may be broken by direct or indirect violence. Direct violence gives rise

to transverse fractures at the same level and such may be directly compounded. Indirect violence accounts for the spiral types at varying levels, and the compounding occurs when a sharp spicule of bone penetrates the skin from within.

Displacement is frequent and overlap occurs from muscular spasm. The oblique fracture is especially prone to interposition of soft parts and is unstable after manipulative reduction.

Clinical Picture.—The nature of the accident determines the clinical picture. Fracture of a single bone causes little alteration in the normal contour of the leg, but involvement of both bones may be accompanied by considerable deformity.

The presence of a surface wound and the degree of soiling must be carefully noted.

X-rays show the type of fracture and presence of comminution.

Treatment.—Fractures of the tibia and fibula are serious injuries because of the time required to secure union. This time factor increases as the line of fracture is placed lower in the tibia. The upper third consists of cancellous bone and is surrounded on two-thirds of its surface with muscles. The blood supply is good to both fragments. The lower third has little muscle in relation to its surface, tendons having arisen to pass into the foot. The bone has a poor blood supply and little cancellous tissue. The middle third is intermediate in this respect. The presence, therefore, of butterfly fragments devoid of circulation in fractures in the lower half indicate a prolonged period before union.

Four methods of treatment are available:

1. Manipulation and plaster cast.
2. Skeletal traction.
3. Open operation with internal fixation
4. External skeletal fixation.

Manipulation and Plaster Cast.—Fracture of the fibula alone requires little treatment apart from protection. The tibia affords adequate splinting.

Fracture of the tibia alone without displacement is best treated in a mid thigh cast with the knee slightly flexed and foot at a right angle.

Fractures of the tibia and fibula without displacement, or those in the young or adult that are transverse and stable after manipulation, can be treated in a similar way.

Internal Fixation.—In cases of fracture of both bones resisting reduction by manipulation or traction, or where compounded, or when both limbs are fractured, or other injuries justify, open operative correction and fixation by screws or plates is used. Post-operative therapy includes protection by plaster cast and ambulation on crutches.

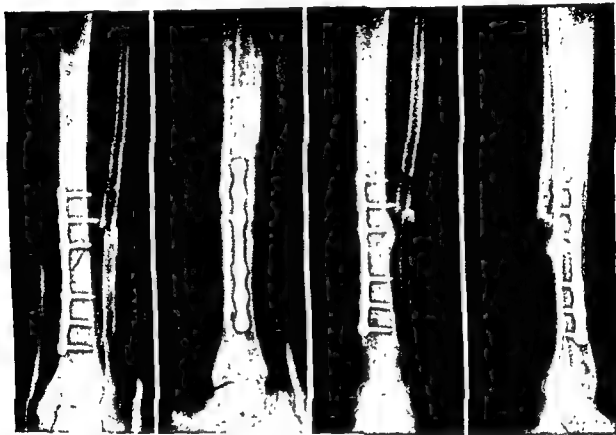


Fig 410—X-rays of fracture of tibia and fibula immediately after plating and three months later, showing clinical union.

Skeletal Traction.—Displaced fractures of the tibia and fibula, especially if oblique or comminuted, can be well treated by skeletal traction through the os calcis on a Braun splint. (See General Section on Fractures.) Preliminary manipulation under anesthesia is used to obtain alignment.

Weights vary from 5 to 12 pounds, and the position is followed by x-rays. After 3 to 4 weeks the limb is placed in a cast from mid thigh to toes, and ambulation on crutches is begun.

External Skeletal Fixation.—This method has been successfully used for displaced fractures of both bones.

Time Factor.—In all cases a period of 12 to 16 weeks is required depending upon the patient's age, and the position of fracture. Fractures in the lower third in the older patient require even longer immobilization.

Complications.—

Delayed Union.—Delayed union is difficult of definition, but in the case of tibial fractures may be said to be present when

movement still persists on changing the cast between 4 and 6 months. When this is found, it is wise to accelerate healing by the application of an onlay bone graft to the anterolateral surface

Nonunion.—When clinical examination after 6 months discloses movement at the site of fracture, and x-rays show sclerosis of the bone ends and minimal callus, union should be assisted by removing the scar tissue and metallic fixation if present. This is followed by application of a sliding or onlay bone graft. Cancellous bone can be placed around the fracture line. Adequate plaster splintage is required postoperatively.

In both delayed and nonunion, general treatment directed to correct dietary or mineral deficiency should be instituted.

Chronic Osteomyelitis—This complication of open fractures involving the tibia, lessened by chemotherapy, is best treated by saucerization of the diseased bone and application of a split thickness skin graft. Cancellous bone may be used later to fill cavities. Plastic procedures supplement the initial treatment.

ANKLE

The ankle is a joint frequently subjected to sprain, fracture, or dislocation. Sprains may give rise to more prolonged disability than fractures if improperly treated.

Sprains

Rupture or avulsion of a few fibers of a ligament does not alter the stability of the related joint and may be called a minor sprain. When the ligament is completely avulsed, instability of the joint results and such injury is best designated a major sprain. This distinction is of the greatest importance in injuries of the ankle with negative x-ray findings.

INVERSION SPRAINS

If the foot is forcibly inverted on the leg, a sprain of the anterior and medial fasciculi of the external lateral ligament occurs. This is the most common ligamentous injury of the ankle. If the sprain is minor, local tenderness is present at the tip of the malleolus or on the tarsal attachments. Strapping immobilization for 7 to 14 days suffices in these cases.

If the sprain is major, there has been a temporary subluxation, and instability of the ankle will result unless the ligaments heal soundly. Clinical diagnosis of the extent of the instability is difficult and requires x-ray studies using local or general anesthesia. When the x-ray is taken in forced inversion, the subluxated position will be seen.

Treatment.—The acute cases require immobilization in a below-knee walking cast for 4 to 6 weeks. The ankle should be at 95° to 100° in neutral position.

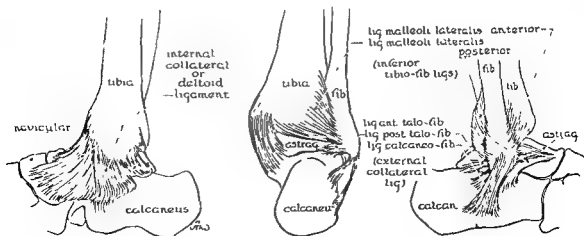


Fig 411—Ligaments of ankle joint.

RECURRENT SUBLUXATION OF THE ANKLE

When the major sprain of the external lateral ligament has been neglected, patients report with a history of repeated "giving way" or "turning" of the ankle. X-ray studies confirm the instability of the joint.

Treatment.—Protective ankle supports may be given a trial. If this measure fails, reconstruction of the ligament with fascia or the peroneus brevis tendon should be considered.

DIASTASIS OF THE INFERIOR TIBIOFIBULAR SYNDESMOSIS

This is the second major sprain involving the ankle that frequently passes undiagnosed. It may be associated with fracture dislocations of the ankle but can occur without demonstrable fracture on x-ray examination.

The injury is produced by a forcible abduction displacement of the foot on the leg. The strong ligaments give way and the fibula springs outward but reduces spontaneously on cessation of the causative force.

Clinically the ankle appears broader than normal and maximum tenderness is localized to the anterior aspect of the tibiofibular joint. Abduction of the foot on the ankle causes considerable pain.

X-ray examination sometimes shows widening of the mortice and increased spread of the tibiofibular joint. A flake of tibia may be avulsed with the ligaments. It is characteristic that the clinical picture suggests a severe ankle injury while the x-ray examination reveals no serious derangement. Local anesthetic injection into the joint followed by x-rays in forced abduction will be the most useful test to confirm the diagnosis.

Treatment.—If correctly diagnosed, prolonged rest (8 to 10 weeks) in a below-knee walking cast with careful compression and molding of the plaster to the malleoli is required. An alternate treatment is internal fixation of the fibula to the tibia by a screw placed above the ankle joint.

Neglect of adequate treatment results in a permanently thickened ankle subject to recurrent pain and swelling.

Fractures and Dislocations

These injuries of the ankle are often grouped together and called Pott's fractures. This corresponds to the designation of fractures and fracture-dislocations of the lower end of the radius as Colles' fractures. It must be remembered that this is a relic of the days before x-rays and that the many varieties of injuries so loosely designated require individual consideration if improvement in standards of treatment is to be obtained.

The classification of ankle injuries follows the writings of Ashurst, who considered them on a mechanistic basis.

Mechanism of Fractures and Fracture-Dislocations.—The foot may be violently displaced on the leg by the following forces:

- (a) External rotation.
- (b) Abduction.
- (c) External rotation and abduction combined.
- (d) Adduction.
- (e) Vertical compression.

Depending on the intensity and duration of the force, different degrees of fracture and dislocation occur. This is best indicated by diagrams taken from type x-rays but it must be remembered that all gradations occur. Further there is frequently an associated compression force when the body weight is transmitted in injuries caused by falls from a height.

Diagnosis.—Fractures without displacement are differentiated from sprains by x-ray examination.

Fractures and dislocations with deformity often present the type appearance expected from the responsible mechanism; e.g., the abducted foot with posterior displacement in the third degree abduction injury. Marked swelling from edema and hematoma

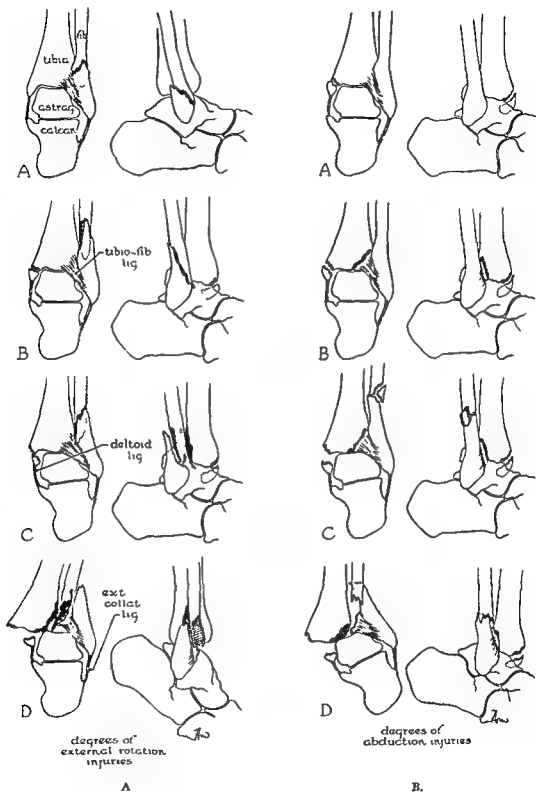
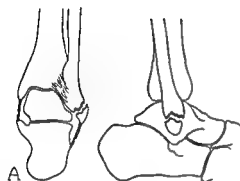


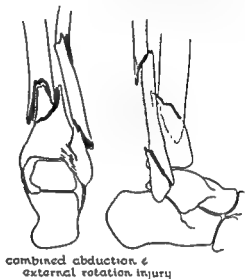
Fig. 412, A and B—Type fractures of ankle

may mask the bony landmarks. X-ray examination with anteroposterior, lateral, and sometimes oblique views is required.

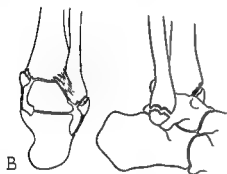
juries with loss of joint stability, this reduction must be maintained until solid healing has occurred in both bony and ligamentous



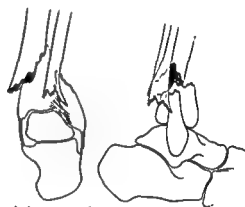
A



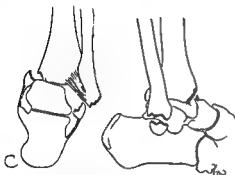
combined abduction &
external rotation injury



B

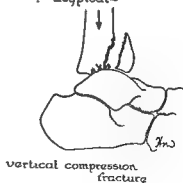


abduction fracture
~atypical~



C

degrees of
adduction injuries



vertical compression
fracture

D.

D.

Fig 412, C and D—Type fractures of ankle.

Treatment.—

Principles.—Fractures and fracture dislocations require exact reduction because this is a weight-bearing joint and the mortice must be properly restored. In the severe in-

juries with loss of joint stability, this reduction must be maintained until solid healing has occurred in both bony and ligamentous structures. It is best to avoid weight-bearing for as long as 6 weeks in these severe cases and plaster casts must be changed as the swelling diminishes and careful molding of the cast around the malleoli is required.

Movements of all muscles under the cast are encouraged to diminish disuse atrophy. Elevation of the limb to prevent edema is important. When the cast is finally removed, support by Unna's paste bandage or elastic stocking is essential, and this should be combined with physiotherapy.

Reduction—The earliest possible reduction of all major displacements is required. This is achieved by reversing the mechanism of the injury. Most posterior displacements are reduced by plantar flexing the foot, drawing downward and then forward as the foot is dorsiflexed. Firm compression of the malleoli by the palms of the hands corrects the widened mortice.

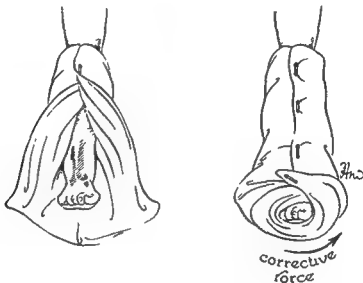


Fig 413—Pillow splint (Gurd's method).

Fracture-separation of the internal malleolus is probably best treated by open operation and internal fixation by screw or suture. This is also true for displaced fractures of the anterior and posterior tibial articular margins.

Fixation.—A below-knee cast, with the foot at 90° (100° in female patients with shortened Achilles' tendon), is the standard method. In undisplaced fractures and those without joint instability, immediate ambulation with weight-bearing is desirable.

If marked swelling is present, the application of a cast can be delayed and the edema reduced by elevation and massage. An alternative is to apply a cast for support, elevate the limb, and change at 7 to 10 days.

In severe fracture-dislocations, a cast is applied, the patient is permitted to be ambulatory on crutches, but weight-bearing is avoided for 6 weeks. A change to a walking cast should be made at this time.

Minor fractures require immobilization for 3 to 4 weeks. Major injuries may require plaster fixation for 11 to 12 weeks.

Most cases require re-education of the limb musculature and arch support on removal of the cast.

Complications.—

1. Traumatic arthritis
2. Post-traumatic edema with thickening of ankle
3. Recurrent dislocation of the ankle
4. Recurrent dislocation of peroneal tendons.
5. Nonunion of internal malleolar fractures
6. Widening of mortice with inferior tibiofibular diastasis

OS CALCIS

Fractures of the os calcis result from a fall on the feet from a height. Isolated injuries may occur from direct violence. The attachment of the tendo achillis may be avulsed by muscular contraction.

Classification.—

(a) Isolated Fractures.

- (1) Posterior tuberosity.
- (2) Avulsion fracture tendo achillis insertion.
- (3) Sustentaculum tali.

(b) Crush Fractures without involvement of subastragaloid or calcaneocuboid joint.

(c) Crush Fractures with involvement of subastragaloid or calcaneocuboid joint.

Clinical Picture.—The patient gives a history of a fall or direct violence to the heel with pain and inability to bear weight on this part.

Examination shows swelling due to edema and hemorrhage. There is acute tenderness on compression.

X-rays should be taken in the standard anteroposterior and lateral planes to include

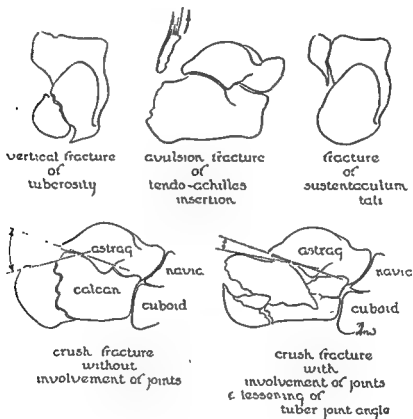


Fig 414—Type fractures of Calcaneus

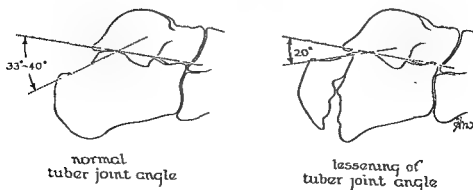


Fig. 415—Lessening of tuber-joint angle.

the ankle. Special axial views are required to demonstrate details of the type of fracture.

In the lateral x-ray, the tuber-joint angle should be estimated to see the degree of upward displacement of the posterior fragments. Attention should also be devoted to the degree of involvement of the subastragaloid and midtarsal joints.

Fractures of the os calcis may be bilateral and are often associated with compression fractures of the dorsolumbar spine. Examination for this related injury should never be omitted.

for this period. The involvement and subsequent arthritis of the subastragaloid and midtarsal joints constitute the main disability after severe injuries and the various treatments do not seem to alter this complication.

Widening of the bone causes impingement on the external malleolus. This is a second complication that appears difficult to avoid. Upward displacement of the posterior fragment results in a relative elongation of the tendo achillis. There is alteration in the cosmetic appearance of the calf and ankle with increased dorsiflexion of the ankle. Finally,

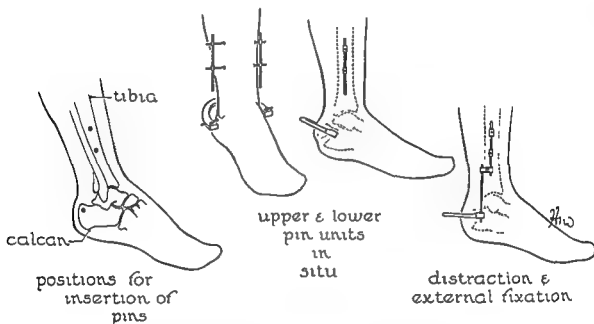


Fig 416—External skeletal fixation for fractured calcaneus

Treatment.—Treatment of isolated fractures without displacement requires little but palliative therapy to diminish pain and swelling. Graduation from crutch to full ambulation may be rapid. Avulsion of the tendo achillis insertion or displaced fracture of the tuberosity requires open operation and suture.

Crush fractures of the body present great difficulties in treatment. It is generally agreed that the cancellous tissue of the os calcis requires 12 to 16 weeks to consolidate and that weight-bearing must be avoided

bony prominences in the sole of the foot may give rise to pain.

These complications, therefore, merit careful consideration of the initial problems presented by the individual case.

The patient is best hospitalized and confined to bed. Compression bandages, hot soaks and massage, associated with elevation of the limb, are used to reduce the swelling. With this is associated repeated manual molding of the bone. After 10 to 14 days, the patient is permitted crutch ambulation without weight-bearing. Some prefer to use

a closely molded cast, but this is not necessary.

In cases of upward displacement of the posterior part and loss of the tuber-joint angle, reduction and maintenance by the two-pin method (Böhler) may be employed. The pins are removed at eight weeks.

After 12 to 16 weeks weight-bearing is permitted. In cases of marked involvement of the subastragaloid joint, arthrodesis is considered 4 to 6 weeks from the original accident. However, this may be delayed to see whether the disability from conservative therapy justifies this procedure.

TARSUS

The *astragalus* is the keystone of the longitudinal arch and constitutes the connecting bone between the leg and foot. Injuries of this bone are uncommon but because of its essential function, the most efficient treatment is required to secure good results

2. Dorsiflexion Injuries.

- (a) Fracture of the neck of the astragalus without displacement.
- (b) Fracture of the neck with subastragaloid dislocation.
- (c) Fracture of the neck with subastragaloid dislocation and backward displacement of the body.

3. Adduction and Abduction Injuries of the Forefoot.

- (a) Midtarsal dislocation with fracture of navicular.
- (b) Tarsometatarsal dislocation with fracture of metatarsals.

Diagnosis.—In cases of complete dislocation of the astragalus, diagnosis is easily made. The injury may be compound. Other cases present typical deformities. The exact post-traumatic anatomy is best assessed by careful x-ray examination.

Treatment.—Reduction should be carried out at the earliest moment. In complete dislocation of the astragalus, especially if

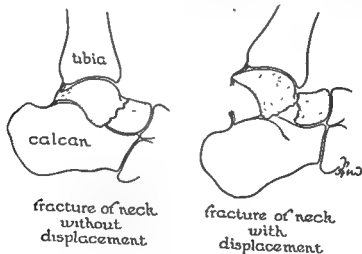


Fig 417—Dorsiflexion fractures of astragalus.

Classification.—Watson-Jones has classified injuries of this bone on the basis of the causative mechanism:

1. Inversion Injuries.

- (a) Dislocation of the ankle.
- (b) Subastragaloid dislocation.
- (c) Total dislocation of the astragalus.

compound, open operative reduction is best. The bone should not be removed even if devoid of ligamentous attachments. In dorsiflexion injuries, the ankle must be fixed in full plantar flexion to maintain reduction.

In severe injuries and dislocations, weight-bearing should be avoided although the pa-

tient can be ambulatory on crutches. Where aseptic necrosis of the astragalus is expected, weight-bearing is contraindicated for 4 to 6 months. The usual case may graduate weight-bearing after 6 weeks. A below-knee cast gives adequate protection

ing the base of the fifth, and necks of the second and third are most common.

BASE OF FIFTH METATARSAL

Fracture in this situation is often called "dancer's fracture." The attachment of



Fig 418—X-ray of Köhler's disease of left navicular with normal right navicular for comparison

Other Tarsal Injuries

Direct violence may cause isolated fractures of other tarsal bones of which the navicular is most commonly involved.

Treatment must be carried out to secure union in satisfactory position, and every effort is directed to preserve the arches of the foot and the muscular balance

Köhler's Disease

Osteochondritis of the navicular occurs from 8 to 13 years and gives pain and tenderness localized to this bone. This is diagnosed by the x-ray appearance; the navicular is narrowed and of increased density.

Metatarsal Fractures

The five metatarsals may be fractured at any point in their length. Fractures involv-

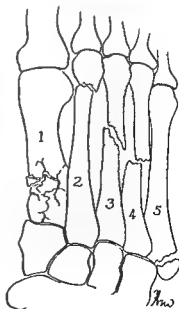


Fig 419.—Composite diagram of type fractures of metatarsals

peroneus brevis is avulsed in sudden inversion of the forefoot. The base may also be fractured by direct violence. Strapping or plaster cast immobilization for 10 to 21 days suffices.

SHAFTS OF METATARSALS

The shafts may be fractured obliquely by indirect torsion or transversely by direct violence.

Treatment.—If displacement is minimal, a walking cast, with sponge rubber molded to preserve the long arch and concavity of the foot, will prove satisfactory. Crutches should be used for 10 to 14 days. The cast is required for 4 to 6 weeks.

If displaced, traction through the corresponding phalanges should secure reduction. Care must be taken to avoid angulation into the sole of the foot. A metatarsal bar will be helpful when walking begins.

NECKS OF METATARSALS

The neck of metatarsal 2 or 3 may fracture from the stress of forced walking. This has been termed "march fracture."

Care must be taken to prevent excessive callus formation, which will give prolonged disability. This is obviated by early recognition and avoidance of unprotected weight-bearing by using a walking cast and crutches for the first 10 to 14 days.

Fractured Phalanges

Crushing injuries of the great toe are the most common. These result from direct injury from falling objects such as carried cases, furniture, etc., or by stubbing the toe. The injury is often compound.

Treatment consists of careful cleansing of the part, evacuation of subungual hematoma, and application of an easily removed splint. Elevation and hot baths when the wounds have closed diminish the swelling.

Ambulation should begin with crutches. A metatarsal bar is applied to the shoe, which has been cut out over the dorsum of the affected toe.

Dorsal dislocation at the first metatarsophalangeal joint is the common dislocation. It is readily reduced by traction in flexion, with direct pressure. The reduction is stable, and protection from the pressure of weight-bearing is the essential point in treatment. The time required is determined by the disappearance of pain and tenderness. Crutches for 2 to 3 weeks is the usual requirement. A metatarsal bar should be applied to the shoe.

COMMON DISORDERS OF THE FOOT

Foot Strain

Painful feet are one of the most common disorders afflicting the human race. The upright posture, the crippling results of improper shoes, and the rigid surfaces of city streets and buildings, all play their part in the causation. It is questionable whether the old Chinese custom of binding female feet is much worse than the fashions and styles in women's footwear of this day.

General Discussion.—Most attention in writing has been devoted to the arches of the feet. These are most important in any consideration of this present problem. Other writers, such as Morton, have stressed the short first metatarsal with subsequent stress strain hypertrophy of the second metatarsal, i.e., Morton's foot. R. I. Harris, in studies of large numbers of feet in recruits, did not find this of great clinical significance.

The writer has found that greatest practical help has resulted from accepting the original structure of the foot as normal for that individual and trying to understand what factors have caused it to break down. If it is remembered that the pain is usually the result of ligamentous strain and if we recall Keith's "Law of the Ligaments," which states the ligaments do not undergo strain until the muscle balance is lost, it is possible to make a physiologic approach to our understanding of the mechanism and its treatment.

Foot strain results from overuse, or improper use, and this may be predisposed to by overweight, by imbalance of the muscles after bed rest, by overuse in forced marching, in work involving carrying heavy weights, or by poorly fitting shoes.

cises to strengthen the tibial muscles will relieve the symptoms. This may be supplemented by the use of a longitudinal arch support and Thomas' heel.

In *chronic foot strain*, the same principles are followed but should be supplemented by



Fig 420—A. Medial view of plaster model of pes planus B. Anterior view showing valgus deformity of forefoot. C. Posterior view showing valgus deformity of heel

The tibial group is weakened in relation to the peroneals and the foot adopts a pronated or valgus position of the forefoot and heel.

In the early stages this is correctible. In *acute foot strain*, protection from weight-bearing, massage, contrast baths and exer-

manipulation of the foot to increase its flexibility.

Correct shoes with well-supported arches and low heels are required. The highly arched foot and the clawfoot (*pes cavus*) require specially built orthopedic shoes if the patient is to secure full use of his feet.

In all cases careful study of the circulation should be made to exclude arteriosclerosis or Buerger's disease as the underlying cause.

Disorders of the Heel

Superficial bursitis occurs over the back of the heel from the friction of slipping shoes. It is especially common when a prominence of bone projects in thin individuals.

Treatment consists in protection from irritation. Sometimes the prominent bone may require resection.

Epiphysitis

Osgood described osteochondritis of the epiphysis of the os calcis. It corresponds to the same process in the tibial tubercle. Diagnosis is by x-ray.

Treatment consists in elevation of the heel of the shoe and protection from sprain or friction during the painful stage in a well-padded, below-knee walking cast.

Tenosynovitis of Achilles' Tendon

This is a common pain syndrome corresponding to tenosynovitis of the wrist extensors. It may result from overuse or strain or may be a rheumatic or gouty manifestation.

Treatment consists in firm support to the area by strapping a felt strip on each side of the tendon. Raising the heel by a sponge rubber pad may help. Salicylates and short-wave diathermy are useful.

Calcaneal Spurs (Plantar Fasciitis)

Severe pain may develop under the heel with a sensation as if the bone were bruised. This is common in overweight individuals from wearing shoes with leather heels and after excessive walking on the city streets. It occurs commonly in those with a gouty or rheumatic tendency. Neisserian infection may be a factor.

Diagnosis is made on the symptomatology and finding the point of maximum tenderness on the plantar surface of the os calcis

X-rays often show a well-formed spur or deposit of calcium. The symptoms bear no relation to the presence or absence of this spur and many believe that we are dealing with a strain of the plantar fascia.

Treatment.—Treatment consists of a sponge rubber support in the heel of the shoe with a hole cut out over the painful area. Attention to overweight, the use of wool socks, and rubber heels, together with avoidance of dietary and alcoholic excesses, will prevent recurrence.

The spur should rarely be resected.

Lesions of Peroneal Tendons

Tenosynovitis and recurring subluxation of these tendons may give rise to pain on the outer side of the heel.

Hallux Valgus

This unsightly deformity is most frequently seen in female subjects and is caused by improper shoes. The inner border of the shoe slopes outward and displaces the great toe, which is thus angulated at the metatarsophalangeal joint. Congenital metatarsus varus predisposes to the disorder.

In the early stages, it is correctable, but when chronic, structural changes make the deformity permanent. Friction of the shoe over the prominent boss of bone on the inner side gives rise to a traumatic bursitis, the so-called *bunion*.

Treatment.—In the early cases, a shoe of proper size and of the Oxford type with cuban heel is advised the female patient for routine use. A toe post may be worn between the great and second toes.

The chronic case is best treated by the Keller operation in which the exostosis on the metatarsal head is removed together with the base of the proximal phalanx.

All cases postoperatively require re-education of the foot musculature and the use of proper shoes.

Hallux Rigidus

This is a painful arthritis of the metatarsophalangeal joint resulting from injury,

often forced hyperextension or stubbing, or from infection. The joint undergoes gradual ankylosis with final rigidity of the great toe. The hallux may be straight or flexed in position. Walking is difficult.

Treatment.—During the early stages a metatarsal bar should be worn. When fully developed an arthroplasty may be performed by resection of the base of the proximal phalanx.

Sesamoiditis

The two large sesamoids under the first metatarsophalangeal joint may be fractured or subject to pressure injury especially in the highly arched foot.



Fig 421 —Hallux valgus

Fracture must be differentiated from congenital bipartite sesamoids by x-ray of both feet for comparison.

Irritation is minimized by protective pads behind the ball of the foot.

When chronically painful, excision must be considered.

Freiberg's Disease

The syndrome involves the second metatarsophalangeal joint. It occurs in cases with the short first and long second metatarsal bones. Stress and strain appear to cause hypertrophy of the bone including its head, which is greatly enlarged. A mechanical arthritis with pain develops. X-ray examination shows the changes. An associated plantar callosity follows.

Treatment consists of resection of the second metatarsal head.

Metatarsalgia

Pain across the transverse arch is a common syndrome. This arch is flattened and the foot broadened. Calluses often exist under the heads of the second and third metatarsals. The wearing of narrow shoes compresses the soft tissues between the bones with subsequent inflammation.

Diagnosis is readily made on the patient's complaints. Compression of the forefoot exaggerates the symptoms.

Treatment consists of rest from weight-bearing, faradism, and active exercises to

the foot musculature, the use of a metatarsal pad to restore the arch, and shoes of proper size and shape.

Morton's Toe

This syndrome is closely related to metatarsalgia. As a result of irritation of the digital nerve between the third and fourth, or fourth and fifth toes, a traumatic neuroma develops. The patient complains of excruciating neuralgia on walking in his shoe, localized in the third or fourth toe, and so severe as to necessitate sitting down on the street and removing the shoe, which gives temporary relief.

Treatment.—When well established, treatment consists of resection of the neuroma through a plantar incision.

Hammer Toe

This is a deformity most commonly seen in the second toe and consisting of hyperextension at the metatarsophalangeal joint and flexion at the interphalangeal joints. The prominent dorsal angulation becomes inflamed from friction and the tip of the toe is irritated by pressure on the sole of the shoe.

Treatment.—Corrective strapping may be tried but is usually ineffective. Tenotomy with arthrodesis of the interphalangeal joint to give a straight toe is the best treatment.

Corns

Corns are epidermal hypertrophic responses to intermittent pressure and friction. They occur over prominences of bone where the shoe rubs. Corns are of two types: (a) hard; (b) soft

The *hard corn* is most commonly seen over the fifth toe, which in some persons is merely a corn-bearing digit. If protection cannot prevent recurrences, removal of the bony phalanges leaving the soft tissues of the toe or ablation of the digit with the head and neck of the fifth metatarsal which narrows the foot, will secure a cure. The chiropodist affords palliative relief in most cases

The *soft corn* occurs between the toes and the underlying cause is a prominent base of phalanx. Excision of this prominence of bone is curative. A pad between the toes also diminishes the pressure

Athlete's Foot

Fungus infection between the toes and on the soles is a frequent disorder in summer months. The characteristic moist desquamating white skin between the toes is well known. Fissures of considerable depth permit entry of pyogenic bacteria and cellulitis.

Prophylaxis.—Careful attention is required to keep the feet clean and dry. The use of rubbing alcohol followed by powder

is efficacious. When secondary infection occurs penicillin ointment followed by undecylenic acid preparations will remove the fungus. Recurrence is common unless appropriate care is given.

Ingrowing Toe Nails

This lesion most frequently affects the nail of the great toe. The etiological factors are tight shoes, tight socks, and improper care of the nail. The pressure of the flesh against the nail edge or corner causes pain. The patient cuts the corner of the nail and the flesh occupies this space. When the nail grows there is no room for it and penetration of the flesh occurs. Thus it is not an ingrowing nail. The nail is of normal width. Rather it is the flesh that has been compressed into this area.

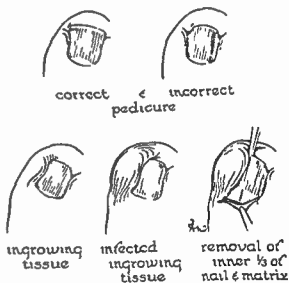


Fig 422.—Ingrowing toe nail.

Prophylaxis.—The edge of the nail and corners should be allowed to grow beyond the flesh and be cut straight across. The corners should never be removed. In the early stages a piece of absorbent cotton under the corner may relieve pain until proper growth has occurred.

Radical.—When seen late with infected granulation tissue, the infection must first be cleared. This may necessitate removal of the nail. If the nail is permanently de-

formed, or if the condition has been recurrent, the nail bed in whole or part may require removal to give permanent relief.

Onychogryphosis

Onychogryphosis is a horny excessive growth of the nail occurring most frequently on the great toe. Some believe that it results from a chronic infective irritation of the matrix. Many cases are due to the nutritional disturbances of a poor arterial circulation.

Treatment.—In the well-developed case, removal of the nail with its matrix is the only satisfactory treatment. This should only be performed when the circulatory status of the limb is adequate.



Fig. 423 —Subungual exostosis

Subungual Exostosis

An exostosis may develop from the terminal phalanx and project forward pushing the nail with it. A very painful condition develops.

Treatment consists in resection of exostosis including its base.

CONGENITAL TALIPES EQUINOVARUS

The word *talipes* is derived from the Latin words, *talus* and *pes*, and is the term used to cover all deformities of the foot centered on the talus. Congenital talipes equinovarus, or clubfoot, is the commonest congenital deformity. It may be unilateral or bilateral; it may occur as the only congenital abnormality or be associated with other stigmata such as cleft palate, spina bifida, arthrogryphosis multiplex, etc. There is a tendency for this defect to run in families.

Etiology.—Various theories as to the causation of this deformity have been postulated. The view most commonly held is that it is due to the persistence of the feet in the plantar flexed and adducted position which is normally present up to the third month of intrauterine life. In recent years experimental work on developing embryos of various animals and birds suggests that congenital defects are developmental arrests which may be due to chemical changes in the fetal environment during the "critical phase" of development. The work of Duraiswami is most interesting in this respect; he has been able to produce most of the typical congenital deformities in chickens by injecting various doses of insulin into the yolk of fertilized eggs. The critical phase of development is different for the various parts of the body. Thus, injection during the first two days of incubation caused defects in the vertebral column, on the third day deformities of the feet, and on the fourth and fifth days deformities of the limbs and beak. A further interesting point shown in this research was that once the tendency to deformity had been induced, the deformities recurred in the next generation. From this research it would appear that dietary and hormonal influences during pregnancy may be the causative factors.

Pathological Anatomy.—Analysis of the deformity shows that it consists of several parts:

1. The foot is plantar flexed. This equinus position takes place at the ankle joint where the talus is angulated plantarward and also at the midtarsal joint where the forefoot is angulated plantarward.

2. The varus deformity occurs mainly at the subastragaloid joint.

the position of imbalance. Walking tends to aggravate the deformity. Pressure points occur from shoes, and where weight-bearing is most marked. In the severe case the patient walks on the outer border of the foot.

Diagnosis.—The clinical picture is so typical that the deformity is usually noted by the medical attendants at birth. When found, the presence of other defects should be suspected. The possibility of a nerve lesion

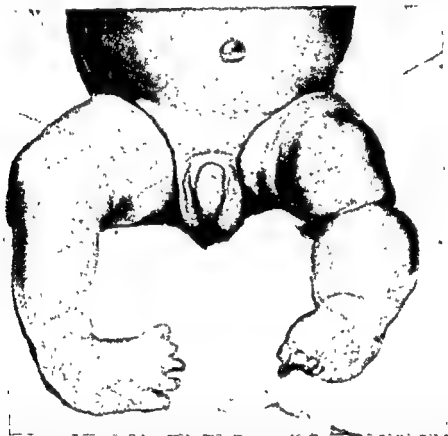


Fig 424.—Photo showing multiple congenital deformities of the lower limbs. On the right side, a talipes equinovarus is present together with failure in the differentiation of the great, second, and third toes. On the left side, there is a congenital constricting band just below the knee. The leg below this is of increased size, and there is a failure of differentiation of the toes.

3. An adduction deformity which is chiefly at the midtarsal joint, which draws the forefoot medially. In the infant this deformity is plastic, but as months and years pass, structural changes increase with alteration in the shapes of the bones, in the structure of the ligaments to fix the deformity, and with contractures of the muscles in

associated with a spina bifida and paralytic type of talipes must be excluded.

Treatment.—The essential points in treatment consist of the earliest possible diagnosis and the immediate institution of corrective measures. Some orthopedists begin treatment at birth; others wait until the child can be brought for treatment.



Fig. 425—Twins presenting a marked degree of bilateral equinovarus, showing the result achieved after orthopedic treatment

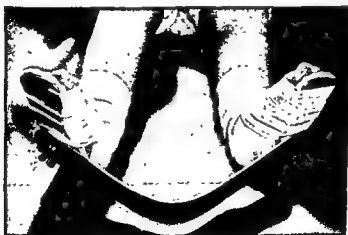


Fig. 426—The Dennis Browne dynamic splinting used on a case of clubfeet. Each foot is first carefully bandaged to a well-padded metal plate and is then fixed, as illustrated, to a metal crossbar. The crossbar is bent so as to secure a valgus position of the foot, and the plate is fixed in external rotation. In this way the kicking movements stretch the shortened soft tissues while developing muscular power.

At this stage the dynamic splinting devised by Dennis Browne is used. This is a method by which the feet are fixed on foot plates which are then attached to a cross-bar which maintains external rotation and valgus. The movements of the limb stretch the shortened tissues while developing the muscle power. Full correction is obtained within a few weeks, but splinting is continued in order to prevent recurrence. When the child commences walking, outside wedges are used on the shoes. A similar type of night splint is applied when the child is resting. Clinical review of progress is continued at intervals until the age of 12 years.

The method of dynamic splinting has replaced repeated manipulation and plaster casts to hold correction which were previously in use.

In those cases which have not received the benefit of early diagnosis and treatment, and which present after the age of 2 years, repeated manipulation and plaster fixation can be employed. It is essential to avoid the use of excessive force. With repeated manipulations at intervals of 3 weeks and plaster fixation, resistant cases can be corrected utilizing the plasticity secured by the osteoporosis of disuse.

In certain of these cases there is a fixed equinus deformity which requires correction by posterior capsulotomy of the ankle joint associated with elongation of tendo achillis by Z-plasty from the medial to lateral side. A further group will require a tibialis anticus transplant to the lateral side of the foot to correct the metatarsus varus deformity.

This is usually carried out at 5 to 10 years of age.

After 11 to 12 years of age, cases presenting appreciable residual deformities are corrected by triple arthrodesis with suitable wedge resections.

REFERENCES

- Ashurst, A. P. C., and Bromer, R. S.: Classification and Mechanism of Fractures of Leg Bones Involving the Ankle, *Arch. Surg.* 4: 51-129, Jan., 1922.
- Böhler, L.: Medullary Nailing of Küntscher, Baltimore, 1948, The Williams & Wilkins Company, pp. 161-248.
- Brockman, E. P.: Congenital Club-Foot (Talipes Equino-varus), New York, 1930, William Wood & Company.
- Browne, D.: Talipes Equino-varus (Arris and Gale lecture), *Lancet* 2: 969, 1934.
- Colonna, Paul: Regional Orthopedic Surgery, Philadelphia, 1950, W. B. Saunders Company.
- Dickson, F. D., and Diveley, R. L.: Functional Disorders of the Foot, Philadelphia, 1939, J. B. Lippincott Company.
- Duraiswami, P. K.: Presentation Before American Academy of Orthopedic Surgeons, Chicago, Jan., 1932.
- Fisher, A. G. Timbrell: Internal Derangements of the Knee-Joint, ed. 2, New York, 1933, H. K. Lewis & Co., Limited.
- Galloway, H. P. H.: Open Operation for Congenital Dislocation of Hip, *J. Orthop. Surg.* 2: 390, 1920.
- Lewin, Philip: The Foot and Ankle, ed. 3, Philadelphia, 1947, Lea & Febiger.
- McMurray, T. P.: Ununited Fractures of the Neck of the Femur, *J. Bone & Joint Surg.* 18: 319-327, 1936.
- Pemister, D. B.: Repair of Bone in the Presence of Aseptic Necrosis Resulting From Fractures, Transplantations, and Vascular Obstruction, *J. Bone & Joint Surg.* 12: 769-787, 1930.
- Smillie, I. S.: Injuries of the Knee Joint, ed. 2, Edinburgh, 1951, & S. Livingstone.
- Smith-Petersen, M. N., Cave, E. F., and Vangorden, G. W.: Intracapsular Fractures of the Neck of the Femur, *Arch. Surg.* 23: 715-759, 1931.
- Steindler, Arthur: Post-Graduate Lectures on Orthopedic Diagnosis and Indications, Springfield, Ill., 1950, Charles C. Thomas, vol. 1.

CHAPTER XL

INFECTIONS OF BONE

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Osteomyelitis is the term used to designate the inflammatory process in bone. When the process is confined to the epiphysis, it is called *epiphysitis*, and when the inflammation is localized to the surface of the bone beneath the periosteum, it is named *periostitis*. The inflammatory process may vary in extent and severity and is classified as acute or chronic in nature.

Bacteria may metastasize to bone in the course of a bacteremia or septicemia associated with a systemic infection resulting in the disease *acute hematogenous osteomyelitis*. In other cases organisms may be directly introduced as a result of trauma which causes open or compound fractures. If the contamination is not adequately removed by surgical débridement, infection of the bone results and this type is called *primary osteomyelitis*. When bacterial infection extends from a neighboring soft tissue inflammatory process such as an abscess or ulcer to involve bone, the type of lesion is termed *secondary osteomyelitis*. The characteristic reaction of bone to the inflammatory process is determined by the rigid nature of osseous tissue and by the arrangement of the blood supply.

The Structure of a Long Bone.—Osseous tissue consists of an organic matrix upon which calcium salts are precipitated to give it a rigid character. A typical long bone comprises a compact bony cylinder called the *diaphysis* which contains the *medullary cavity*. At each end there is an *epiphysis* of cancellous bone covered by articular cartilage which enters into the formation of the neighboring articulation. Between the epiphysis and diaphysis in the maturing individual lies the area of maximum growth called the *epiphyseal plate*. The diaphyseal side of this epiphyseal plate receives the richest blood

supply. In this area the nutrient vessels end in large capillary loops, and bacteria carried by the blood stream tend to stagnate there. Besides, in the young patient, twisting strains find in this area the weakest point in the bone-joint-structure as is well known from epiphyseal fracture-separations. This area of the bone so important in growth, injuries, and infections, is named the *metaphysis*.

The Blood Supply of a Long Bone.—The work of Lexer remains the classic study in this field and the arrangement can be understood by reference to the blood supply of the tibia. The main nutrient artery enters on the posterior surface of the upper third of the tibia and divides into an ascending and a descending branch which proceed to the respective epiphyseal plates. The branches of the artery pass longitudinally in the medullary canal and in the compact bone through the longitudinal or Haversian canals which contain in addition the nerves and lymphatics. Besides the arterial circulation to the diaphysis, a second supply derived from the periosteal circulation enters the compact bone along the Volkmann's canals which run tangentially to the surface. At each end of the bone, arteries enter the epiphysis separately and enter the metaphysis from the rich arterial anastomosis around the joint. Because of the arrangement of the vessels in rigid canals, any inflammatory process in bone tends to raise the tension around the vessels and soon causes occlusion and thrombosis. The inflammatory processes within the bone soon release their tension by spread along Volkmann's canals to the subperiosteal area. This extension by stripping off the periosteum interrupts the periosteal circulation and again leads to interference with the nutrition of bone and produces local death of tissues.

The Reaction of Bone to Infection.—The main effect of the inflammatory process on bone is the interference with its nutrition by occlusion and thrombosis of the blood supply. Bacterial toxins also play their part in destroying the living bone cells or osteoblasts. This local destruction of bone is called *necrosis*. If an area of bone has undergone necrosis, the adjacent living tissue reacts to separate and extrude the dead portion. When the area of necrosed bone has been separated, it is called a *sequestrum*.

In flat bones such as the skull and pelvis, the reaction to the infection differs from that in long bones and corresponds more to the cellular death of tissue seen in soft tissue ulceration. This process in bone is called *caries*. Most studies of the progress of infection in bone are made by repeated radiological examinations. During the early inflammatory process, decalcification due to the intense hyperemia causes a loss in the normal density of the x-ray shadow. This change in bone density and radiological appearance is

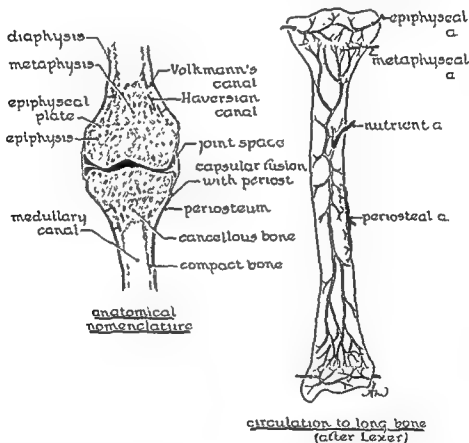


Fig. 427.—The circulation in a long bone

While this process of separation and extrusion is taking place, the periosteal area of bone proliferates to give strength to the bone as a whole and produces a cylinder of new bone around the damaged area. This new cylinder of repair bone is called the *involucrum*. The area of sequestration sooner or later communicates with the exterior by *cloacae* traversing this involucrum and it is through these sinuses that nature attempts to extrude the sequestrum

referred to as *osteoporosis*. The increased density which follows ischemia of bone and repair with scarring is termed *osteosclerosis*.

ACUTE HEMATOGENOUS OSTEOMYELITIS

Before the advent of penicillin and the other antibiotic agents, few infective processes afforded the surgeon greater difficulty in treatment than acute hematogenous osteomyelitis. In the fulminating type, death

from the septicemia and generalized pyemic abscesses throughout the body was of frequent occurrence. In cases of lesser systemic severity, the extensive necrosis of bone with subsequent sequestration, sinuses, recurrent abscesses, and adjacent joint ankylosis, often left the patient a cripple for life. Today, with early diagnosis and appropriate

antibiotic therapy, the systemic and local processes can be rapidly controlled, and a dramatic improvement in the mortality and morbidity rates has resulted.

Bacteriology.—The vast majority of cases of acute osteomyelitis (90%) is caused by *Staphylococcus pyogenes*. A relatively small number is due to *Streptococcus pyogenes* or *D. pneumoniae* and rare individual cases may be caused by other microbes. In acute hematogenous osteomyelitis the causative organism can be isolated from the bloodstream or the local lesion in pure cultures. In primary or secondary osteomyelitis mixed infections may be found and have to be treated accordingly. *Streptococcus pyogenes* and *D. pneumoniae* infections are more prone to involve the epiphysis and the neighboring joint.

Pathogenesis.—The most common source of staphylococcal osteomyelitis is an infection of the skin, furuncle, or carbuncle, etc. From the cutaneous lesion invasion of the bloodstream occurs, with or without clinical manifestations, and the organisms are carried to the metaphysis of the bone. The most common site involved is the region of the knee including the upper end of the tibia and lower end of the femur. The ankle is second in frequency and the metastasis involves the lower end of the tibia or lower end of fibula. The upper end of the humerus and upper end of the femur are also common sites, but it must be remembered that any metaphyseal area may be involved. Re-entry of bacteria from the local lesion into the circulation may in turn cause septicemia or further metastatic foci in other bones or organs.

Various factors predispose to the development of this disease. Among these the age of the patient is most important. The age period marking the maximum incidence is 5 to 12 years but all ages from the first week of life until epiphyseal closure around 20 years may be affected. Boys are more commonly afflicted than girls and this sex incidence lends support to the theory that juxta-epiphyseal strain caused by an injury is a



Fig. 428.—Photograph of museum specimen of chronic osteomyelitis of the tibia showing involucrum, sequestrum and cloacae.

predisposing factor. As previously mentioned the area of the metaphysis is the weak link in the bone joint structure in the growing patient and strains produce a hematoma affording a nidus for circulating bacteria. *Lowered resistance and climatic influences* are often mentioned as contributing factors. Cases of osteomyelitis are more common in the late winter months in the north temperate climates and in the poorer classes of the population.

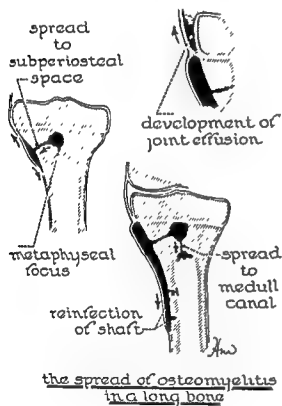


Fig 429.—The spread of osteomyelitis in a long bone.

The Pathological Spread in Bone.—The accepted ideas on the spread of the infection from the fixation point in the metaphysis follow the observations of Starr and are summarized in the accompanying diagram. As the tension rises from the suppurative process, the inflammation extends into the cancellous tissue and down the medullary canal. The next extension is outward to the subperiosteal space along the Volkmann canals. The epiphyseal plate affords a strong

barrier to the passage of bacteria into the epiphysis and thus into the joint. Once the subperiosteal space is involved, the tension again rises and the periosteum is stripped off the bone and the shaft infected via Volkmann's canals. Because of the close relation of the subperiosteal abscess to the synovial reflexion and joint capsule, an effusion into the joint may occur, and in time a pyogenic arthritis develops in the neglected cases.

In certain cases the embolism may affect the metaphyseal area only; in other cases the embolus may block either the main ascending or the main descending branch of the nutrient artery or the entire vessel may be blocked in the initial stage. These different sites of embolism determine the pattern of the inflammatory process and have been emphasized by Wilensky.

As the bone is rendered ischemic by the thrombophlebitis and also by the cellular death caused by bacterial toxins, the process of sequestration begins. In the neglected cases in the preantibiotic era the separation was completed in the average case in 6 to 8 weeks.

Clinical Types of Acute Hematogenous Osteomyelitis.—There are three main clinical types of this disease determined by the severity of the systemic and local process:

1. The fulminating type in which septicemia and pyemia predominate and the bone lesion is of secondary importance.
2. The type where the bloodstream invasion and osteomyelitic process are practically silent. The bacteremia passes unnoticed and the osteomyelitis becomes clinically apparent with the development of a subacute or chronic Brodie's abscess.

3. The common type where a septicemia of varying severity exists for several days and the osteomyelitic process predominates.

Clinical Picture.—The clinical picture will vary depending on the intensity of the septicemic and osteomyelitic processes.

In the fulminating type the young patient is desperately ill and frequently delirious and irrational. The temperature ranges above

104° F, the pulse is 120-160, and the leukocyte count 25,000 or more. The patient is often dehydrated. Complaints may be of fleeting pains from one bone or joint to another. The general symptoms and signs far overshadow any local signs in bone which may not be discovered. A blood culture is usually positive if taken just before the peak of the temperature and the prognosis can often be judged by the number of colonies cultured and the number of days the septicemia persists. Before the advent of penicillin the majority of these patients died from generalized pyemic abscesses in brain, lung, kidney and other organs.

of function of the neighboring joint. In the lower limb a limp may be noted or a tender enlargement of the bone.

In the *common type* of case with a septicemia of several days' duration and an acute inflammatory process in one of the long bones, the symptoms and signs will be both general and local.

The general features will be determined by the intensity and duration of the septicemia. The patient will be ill with a raised septic type of temperature, rapid pulse rate, and leukocytosis. Repeated blood cultures in the early stages will be positive and enable the isolation of the organism and study of



Fig. 430—X-ray showing early changes of acute hematogenous osteomyelitis.

A Primary lesion

B Several weeks after evacuation of subperiosteal abscess with primary closure and antibiotic therapy. Note decalcification and periosteal thickening.

In the *silent type* with the development of a Brodie's abscess, the general symptoms and signs are negligible. The patient complains of an increasing pain or aching at the extremity of a long bone with some limitation

of the antibiotic sensitivity. As previously stated 90% or more are due to the staphylococcus.

The local symptoms are those of a circumscribed acute infection. The patient com-

plaints of an increasing intense and throbbing pain situated deep in the limb which will be localized by the examiner on palpating the area of maximum tenderness. The area may be swollen and reddened. If the process is of several days' duration, a subperiosteal abscess with fluctuation may be present.

Radiological examination in the early stages is generally negative but an area of rarefaction near the epiphyseal line will be apparent after 7 or more days.

Differential Diagnosis.—The history of a sudden illness with acute pain in one of the limbs in a young active child or adolescent should suggest an acute hematogenous osteomyelitis. The diagnosis will be assisted if there has been a recent skin or upper respiratory infection. In the infant an infected umbilicus should be looked for. Acute osteomyelitis must be differentiated from acute rheumatism. In *acute rheumatism* the pain, tenderness and swelling around a joint usually appear at once, whereas in acute osteomyelitis the pain generally precedes the swelling by two or three days. Besides, in acute rheumatism it is customary to have the pains in several joints and often fleeting from one to another. It is best to regard any acute process at the end of a long bone in the child or adolescent as acute hematogenous osteomyelitis and treat promptly as such by appropriate antibiotic therapy. If the process is rheumatic in character, the response to antibiotics will not be satisfactory but will usually be dramatic when salicylates are administered.

In *poliomyelitis*, the young patient may complain and give signs of pain and loss of function in one limb but characteristically the patient is not so ill nor the local signs so marked as in acute osteomyelitis. Neck rigidity will be present and lumbar puncture will reveal increased cells in the cerebrospinal fluid.

The differentiation from a *sprain or fracture* may be required but in these cases the systemic signs of an infection are lacking. However, the association of juxtaepiphyseal

injuries with osteomyelitis must be remembered. Radiological examination often discloses minor degrees of epiphyseal injuries with flakes of bone torn off with the periosteum or fragments of the metaphysis fractured.

Treatment.—The treatment of acute hematogenous osteomyelitis had undergone great changes by the time the chemotherapeutic and antibiotic drugs had arrived. More and more, surgeons were devoting their attention to supportive general treatment directed toward the septicemia and treating the local process in bone with rest to the part and masterly inactivity from an operative point of view. The results of radical removal of cortical bone and exposure of the medullary canal had not been satisfactory and many surgeons were content to open the periosteum and place fine drill holes into the metaphyseal area to lessen the tension and afford drainage. The use of the antibiotic drugs has made the treatment of acute osteomyelitis practically a medical problem.

The principles underlying the modern treatment may be summarized as follows:

- 1 The patient should be treated in bed and general supportive measures given. Small transfusions are valuable in the early and late stages.

- 2 Blood cultures should be taken to isolate the specific organism and study its antibiotic sensitivity.

- 3 While awaiting the results of blood culture, penicillin in massive doses and streptomycin should be given. In the fulminating type intravenous penicillin should be administered. If the organism does not respond promptly to this regimen, aureomycin should be tried in place of penicillin.

- 4 The bone involved should be placed at rest in a plaster of Paris splint with immobilization of the adjacent joints. Should the case be seen late and a subperiosteal abscess be present, bacteriological study of the aspirated pus should be made. The subperiosteal area may be irrigated and local peni-

cillin, 300,000 units, instilled. Similarly, if an effusion into the joint is present, it should be aspirated and the fluid cultured. If bacteria are present, penicillin should be instilled.

It may be mentioned that there are those today who believe that the local treatment should include the incision of the periosteum and washing out of the subperiosteal abscess and primary closure of the wound. Others prefer to add to this multiple drill holes into the metaphyseal area to diminish the tension. More and more, the cases are being diagnosed and treated at an earlier stage. It can almost be stated that if prompt treatment by the appropriate antibiotic is given within 5 days of the onset, the general and local processes can be controlled. After this period there is a greater incidence of bone necrosis leading to sequestration.

Complications.—In the fulminating cases, toxemia and pyemia with multiple metastatic abscesses in the organs and serous cavities lead to death.

In the cases which are neglected, sequestration with the development of recurrent abscesses and sinuses are the clinical features of one type of chronic osteomyelitis. Such cases so frequent in the pre-antibiotic era should soon become uncommon.

CHRONIC OSTEOMYELITIS

Patients presenting chronic osteomyelitis include the end results of three groups of cases in which the acute primary infection has not been eradicated and the bacteria have become organized in the scarred and devascularized bone. The radiological examination shows osteosclerosis and probably sequestration.

These three groups of cases are:

1. Late cases of acute hematogenous osteomyelitis
2. Open fractures including those due to war wounds with presence of foreign bodies, shell fragments, etc.
3. Chronically infected bone from spread from adjacent soft tissue infections

The cases of chronic osteomyelitis following each of these primary types of infection are becoming less frequently seen because of the prompt and efficient antibiotic therapy in the early stages but during World War II countless numbers of Group 2 were seen and great advances made in their treatment. The time has come when the severe general reaction to such chronic infections with weight loss, secondary anemia, lowered plasma proteins and amyloid disease is rarely seen.

The principles underlying the local treatment of the infected bone are based on the fact that the bacteria are protected from antibiotic drugs by the scarred and devascularized bone. It is therefore necessary to remove such bone, scarred and adherent skin and soft tissue together with any sequestra and foreign bodies that may be present. This is done after the general health has been improved by appropriate transfusions, nutritional and vitamin therapy as is required, and under the protection of antibiotics begun 3 to 5 days preoperatively. A careful bacteriological study of the wounds is made prior to operation and the nature of the mixed infection is determined. Where possible the local condition is improved by topical application of antibiotics prior to operation.

Once the dead bone and scarred tissues have been removed, the wound is closed with a petrolatum pack for 7 to 10 days. In some cases the surgical procedure results in the saucerization of a long bone, in others an exteriorization of a cavity. At the end of this period if a clean granulating surface is provided, the area is covered with a split thickness skin graft. Small sequestra may separate through such a covering, but when the skin graft has finally closed the area, consideration may be given to filling defects by various procedures, which include the use of cancellous bone chips to fill cavities, and the transfer of muscle, fat and skin flaps to improve the cosmetic appearance.

By such means and with the protection of systemic and local antibiotic therapy, many limbs previously amputated and others presenting recurrent abscess and sinus formation, can now be cleared of their chronic osteomyelitis.

charge pure cultures of typhoid bacilli and can cause contamination of other patients.

Tuberculous Osteomyelitis is also today of decreasing significance. Usually the initial lesion is in the juxta-articular area and involves the corresponding joint. This has



Fig. 431—X-ray showing late changes of chronic osteomyelitis

SPECIFIC TYPES OF OSTEOMYELITIS

Typhoid Osteomyelitis is now a rare condition due to the public health measures controlling the water and milk supplies. It is mentioned to remind the student that in typhoid cases involvement of bones such as the tibia, rib, sternum, and vertebrae may occur in the 2nd or 3rd week of the disease or even years after the disease has subsided. In these cases the abscesses if incised will dis-

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Syphilitic infection of bone is also a relatively rare condition from the surgical stand-

point. Efficient and early treatment of the initial lesion in the free clinics has caused the disappearance of such cases.

REFERENCES

- Altemeier, W. A., and Helmsworth, J. A.: Penicillin Therapy in Acute Osteomyelitis, *Surg., Gynec. & Obst.* 81: 138-157, 1945
- Altemeier, W. A.: Treatment of Acute Hematogenous Osteomyelitis With Penicillin, *Ohio State M. J.* 42: 489-496, 1946
- Buchman, J.: Rationale of the Treatment of Chronic Osteomyelitis, *Bull. Hosp. Joint Dis.* 9: 177-181, 1948
- Buchman, J., and Blair, J. E.: Precautionary Administration of Penicillin in Surgical Procedures on Bones and Joints, *Arch. Surg.* 55: 743-750, 1947.
- Buchman, J., et al.: Surgical Management of Chronic Osteomyelitis by Saucerization, Primary Closure and Antibiotic Control, *J. Bone & Joint Surg.* 33: A 107-118, 1951.
- Caldwell, G. A., and Wickstrom, J.: Closed Treatment of Acute Hematogenous Osteomyelitis, *Ann. Surg.* 131: 734-742, 1950
- Dickson, F. D.: *Diagnosis and Treatment of Acute Hematogenous Osteomyelitis*, Chicago M. Soc. Bull. 50: 78-84, Aug., 1947.
- Lewin, P., et al.: Osteomyelitis, *S. Clin. North America* 27: 183-207, Feb., 1947.
- Lexer, Erich, and Kuliga, P., et al.: Untersuchungen über Knochenarterien mittelst Röntgenaufnahmen injizierter Knochen und ihre Bedeutung für einzelne pathologische Vorgänge am Knochensystem, Berlin, 1904, A. Hirschwald, p. 23.
- Orr, H. W.: The Treatment of Osteomyelitis and Other Infected Wounds by Drainage and Rest, *Surg., Gynec. & Obst.* 45: 446-464, 1927.
- Orr, H. W.: Osteomyelitis and Compound Fractures and Other Infected Wounds, Treatment by the Method of Drainage and Rest, St. Louis, 1929, The C. V. Mosby Company
- Robertson, D. E.: Acute Hematogenous Osteomyelitis, *J. Bone & Joint Surg.* 9: 8-23, 1927
- Royal Society of Medicine: Treatment of Acute Osteomyelitis With Penicillin, *Lancet*, pp 236-237, June 15, 1946.
- Starr, C. L.: Acute Hematogenous Osteomyelitis, *Arch. Surg.* 4: 567-587, 1922.

CHAPTER XLI

TUMORS OF BONES AND JOINTS

J. GORDON PETRIE, M.D.

Tumors of the bone may be benign or malignant. In addition metastatic tumors of bones arise from malignant growths in other parts of the body.

GENERAL CONSIDERATIONS

The *benign bone tumors* grow slowly, do not metastasize, and rarely show abnormal blood findings.

The *primary malignant bone tumors* vary in rate of growth, do metastasize, and frequently show blood changes to help in differentiation.

The *metastatic tumors* to bone are most frequently from carcinoma of the breast, prostate, kidney, thyroid and lung.

The exact cause of bone tumors is unknown. Trauma is usually considered an important factor in the development of certain bone tumors.

The patient suspected of a bone tumor should have a careful history, a complete general examination, a hemogram, urinalysis,

blood serologic and chemical determinations, a roentgenogram and a biopsy.

Laboratory Examination.—Certain types of bone tumors produce changes in the blood and urine which may help in their differentiation. Alkaline phosphatase is secreted by proliferating cartilage cells and osteoblasts and excreted by the liver, so there is an increase of alkaline phosphatase in the blood with liver impairment, and also during the active phase of bone growth. This is found also in the healing of fractures and in certain bone tumors such as osteogenic sarcoma and in patients with bone metastases especially from the prostate.

Acid phosphatase is an enzyme found in large amounts in hypertrophy or in carcinoma of the prostate gland. When the carcinomatous tumor has ruptured the capsule of the gland and metastasized to the soft parts or to the bones, there is, in about 70% of cases, an increase of acid phosphatase in the circulating blood.

TABLE XIX
CLASSIFICATION OF BONE TUMORS

| TYPE | BENIGN | MALIGNANT |
|-------------------------|---|---|
| (1) Osteogenic Series | (a) Exostosis, Osteoma (b) Osteoid Osteoma | Osteogenic Sarcoma (a) Medullary and Subperiosteal (b) Telangiectatic (c) Sclerosing Periosteal Sarcoma Fibrosarcoma (a) Medullary (b) Periosteal Capsular and Periosteal Sarcoma |
| (2) Chondroma Series | Chondroma Myxoma | Chondrosarcoma Myxosarcoma |
| (3) Giant Cell Series | Benign Giant Cell Tumors | Malignant Giant Cell Tumors |
| (4) Angioma Series | Hemangioma | Angiosarcoma Angioendothelioma |
| (5) Reticuloendothelial | Solitary Myeloma | (a) Reticulum Cell Sarcoma (b) Ewing's Sarcoma (endothelioma) (c) Multiple Myeloma (d) Myelosarcoma |
| (6) Fatty Tissue Series | Lipoma | Liposarcoma |

The Bence Jones protein may be found in the urine in 60% of cases of multiple myeloma. Bone marrow biopsy from the sternum is positive in 95% of cases of multiple myeloma.

In all bone tumors where there is any doubt as to diagnosis, a biopsy should be done. A biopsy must always be performed before an amputation is carried out on a patient with any bone tumor. The wound after surgical biopsy must always be closed and never packed or drained.

BENIGN TUMORS

Osteoma

Osteomas or exostoses frequently arise from a bony surface. Those arising from the center of the bone are known as enostoses. An osteoma may be compact or cancellous. The former frequently originate from the periosteum of membranous bones, e.g., skull or face. Treatment by excision is indicated when such a tumor causes pressure on nerves or deformity. A large group of exostoses

originate at the ends of long bones, are often multiple, hereditary, and associated with other abnormalities.

Osteoid Osteoma

This tumor, described by Jaffe in 1935, occurs most frequently in patients between 10 to 25 years of age. In general, long bones are affected. There is slight swelling and localized tenderness, but no heat or redness. The roentgenogram shows a zone of sclerosis, in which is an area of rarefaction and a nidus. Either sex may have the tumor. The differential diagnosis includes Brodie's abscess, Garré's sclerosing osteomyelitis, eosinophilic granuloma, Paget's disease and fibrous dysplasia. The differentiation may be done by the history, the x-ray and at times the biopsy. The treatment is surgical excision of the nidus; if removal is incomplete, the tumor continues to grow and to cause symptoms. Histologically, one sees cellular proliferation (osteoblasts), patches of osteoid tissue and areas of calcified atypical bone.



Fig 432.—Bilateral multiple osteochondromas of lower end of femur, and upper end of tibia.

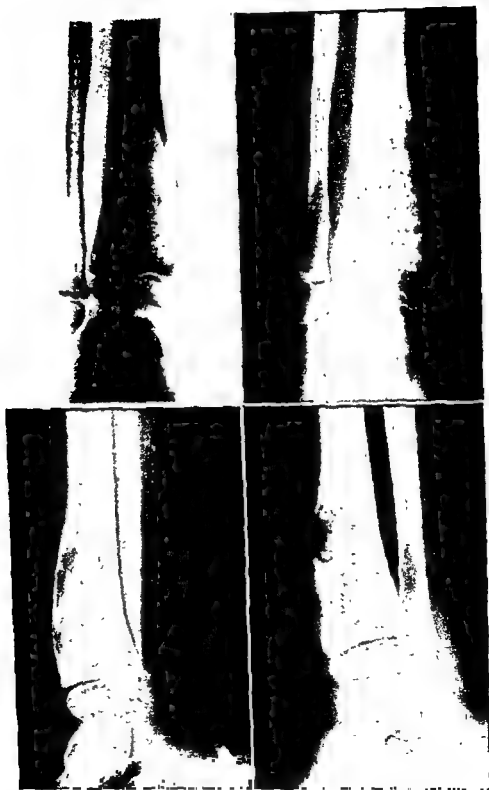


Fig. 433—Giant cell tumor, lower end of tibia, before and after operative excision, and filling of cavity with cancellous bone chips.

Osteochondroma

This common tumor occurs chiefly between 10 and 30 years of age and is most frequently found in the region of the knee joint. The symptoms are usually associated with interference of function of the joint or tendons. Surgical excision should be carried out. These tumors are often encapsulated and can be readily shelled out. Degenerative changes occur so that myxomatous degeneration, cyst formation, calcification and ossification are frequently seen. Geschickter and Copeland state that malignant transformation occurs in 7% of cases. The growth then becomes much softer, the regularity of cell arrangement is lost and invasion occurs.

Chondroma

This tumor occurs centrally (enchondroma) in the small bones of the hands or feet or projects from the surface of the bone (exochondroma). The centrally occurring chondromas cause expansion of the bone and are best treated by curettage and by filling the defect with bone chips.

Giant Cell Tumor—(Osteoclastoma)

This tumor occurs between 15 and 30 years of age. The lesion is locally destructive and tends to recur but does not metastasize. The commoner locations are the lower end of the femur, the upper end of the tibia, and the lower end of the radius. They may also occur in the jaw, pelvis, tendon sheaths, bursae, and capsules of joints. The tumor arises in the epiphyseal area of the bone as a soft, dark-red, hemorrhagic mass, which is frequently lobulated and causes expansion. Periosteal new bone does not occur unless a pathological fracture has been present.

Appearance of the Tumor.—The tumor is cystic, hemorrhagic and traversed by bony trabeculae. The cortex is extremely thin. On microscopic section it shows 3 types of cells:

1. Small round cells which are numerous

2. Large multinucleated (15-20 nuclei) giant cells.

3. Spindle cells

The stroma shows spindle cells and also vascular spaces. The treatment of choice is curettage and insertion of bone chips unless the tumor occurs in a bone where resection is possible. Curettage must be complete, as recurrences follow those incompletely treated. If a recurrence follows, then a thorough curettage should again be done and the cavity filled with bone chips, or, if possible, excision of the tumor and replacement of the defect with bone grafts and bone chips.

Roentgen therapy should be used only on inaccessible tumors or when excision would lead to mutilation or functional impairment.

Some persons doubt that there is such a tumor as a malignant giant cell tumor.

Hemangioma

This occurs usually in the spine or in the skull. In the roentgenogram of the vertebra the appearance is honeycombed and may show collapse and wedging of the body. It must be distinguished from an ordinary compression fracture. This tumor responds to roentgen therapy. Occasionally it occurs in a long bone.

Solitary Cyst of Bone

Solitary cysts of bone occur typically in the metaphysis and adjacent diaphysis of long bones before the related epiphysis has closed and thus usually before the age of 20 years. The x-ray appearances are similar to those of giant cell tumor but this usually occurs after the age of 20 and involves the epiphysis itself. Some authorities believe that the two conditions are closely interrelated.

The solitary cyst may be simple or multilocular. The exact causation is unknown but the cyst may arise as a result of a metaphyseal hemorrhage or as the result of an attenuated central bone abscess.

The patient seeks help most frequently for a fracture which has occurred through the cyst. The common sites of involvement are the upper end of the humerus and upper end of the femur.

Treatment.—A considerable proportion (c. 50%) are cured by the healing process after the pathological fracture. If the cystic changes persist or extend after fracture or are found on x-ray examination for pain, operative treatment is indicated. This consists of removal of an adequate area of compact bone to expose the cavity, thorough curettage of the wall, and cauterization with saturated solution of zinc chloride followed by packing with cancellous bone chips. Protection of the area by immobilization in plaster is required until the x-rays show consolidation. X-ray treatment is best avoided because of the possibility of damage to the epiphyseal plate.

PRIMARY MALIGNANT TUMORS OF BONE

These are found chiefly in the younger age groups. They are relatively rare in comparison with other malignant growths and can present many difficulties in diagnosis to the roentgenologist, pathologist, and surgeon. Successful treatment of bone sarcoma, as in other types of malignant lesions, depends upon early diagnosis, the type of tumor, and to some extent on its location

Osteogenic Sarcoma

This is the most malignant and most common bone tumor. Mayo Clinic statistics show that in 75% of cases, osteogenic sarcoma occurs about the knee. Most cases occur between 10 and 20 years of age and slightly more in males than in females. The metaphysis of the lower end of the femur or that of the upper end of the tibia is usually involved, other sites being upper end of humerus, upper end of fibula, pelvis and scapula

Symptoms and Signs.—Pain, at first intermittent and then continuous, is the chief symptom. It may be accompanied by a limp and later swelling may be seen. The diagnosis must take into consideration many factors, history, physical examination, roentgenogram of tumor and of chest, laboratory examination, surgical biopsy, and previous treatment. Microscopic examination of the tissue is the most valuable aid but this may also be difficult if the tumor has been previously irradiated, or if infection has occurred from a previous biopsy. Laboratory examinations should include urinalysis, hemogram, and blood Wassermann. The alkaline phosphatase is increased in osteogenic sarcoma. Osteogenic sarcoma metastasizes by the blood stream to the lungs and to other bones.

Histology.—The microscopic picture is most variable in this lesion. Spindle cells predominate especially in the more malignant types and occasional giant cells are present. The intercellular substance may consist of mixtures of cartilaginous, myxomatous, osteoid, osseous, fibrous, and hyaline tissue

X-ray Features.—These vary somewhat depending whether the tumor is osteolytic or osteogenic. Early, the x-rays show an area of bone destruction in the subcortical zone extending outward. Strands of new bone formation are seen with raising of the periosteum, but no expansion of the cortex. The new bone is frequently laid down as radiating spicules at right angles to the shaft.

Treatment.—Amputation is usually the preferred treatment. Partial amputation or resection has been considered for some cartilaginous tumors in the distal end of an extremity. Osteogenic sarcoma in children may be radiosensitive and x-ray may be used as a preoperative treatment. X-rays of the lungs should always be taken before surgical removal is undertaken.

Site of Amputation.—One must decide whether to do the amputation through the

bone affected above the tumor, or through the bone proximal to the bone affected. When amputation is through the bone affected above the tumor, a section should be made from the end of the bone removed, and if malignancy is present, amputation above the joint or disarticulation is indi-

Treatment.—Biopsy should first be done. If the diagnosis is confirmed, early amputation is necessary.

A secondary type of chondrosarcoma may arise from a pre-existing osteoma, chondroma, or Paget's disease. Biopsy for confirmation should be performed. If micros-



Fig 434—Osteogenic sarcoma involving the upper shaft of femur, showing the radiating spicules of bone and raised periosteum in anteroposterior and lateral views

cated. If pulmonary metastases are already present, no surgery should be done, unless for palliative reasons, e.g., to remove a useless leg or for pain.

Chondrosarcoma

This tumor appears in adolescence and early adult life. It occurs chiefly at the lower end of the femur and upper end of the tibia. The tumor consists of cartilage, connective tissue, and bone derived from malignant tumor cells.

copy discovers sarcoma, amputation is justifiable in the absence of pulmonary metastases.

Endothelial Myeloma (Ewing's Sarcoma)

This tumor is found in children and in young adults. The shafts of long bones are most commonly involved, but occasionally the jaw and small bones are also affected.

Fever is a frequent sign of Ewing's tumor and not of other bone tumors. The differentiation between this tumor and osteomye-

litis has always been difficult but is more so if antibiotic or preliminary x-ray therapy has already been given. The sedimentation rate may be raised in these cases, but the leukocyte count is not usually as high as in cases of osteomyelitis.

The diagnosis is to be made by biopsy and microscopic examination.

The structure of the tumor is semisolid and of a greyish white color with areas of hemorrhage. The tumor extends along the medullary canal as well as through the Haversian canals to the surface of the bone. The inner layers of bone are destroyed while new layers of bone are deposited on the outside, described as "onion layers" in the x-ray picture.

The common sites are the shafts of the long bones (tibia, humerus, femur, fibula). The os calcis is the most frequently involved small bone.

Histology.—Small round and polyhedral cells are arranged in solid masses. The intercellular stroma is scanty. The picture may be obscured by degenerative changes or previous irradiation.

X-ray Features.—Central rarefaction of a patchy type with sclerosis of the cortex, and "onion peel" new bone is characteristic.

Treatment.—This is one of the most radiosensitive of all tumors, and x-ray therapy should always be given after the diagnosis has been established. This usually results in diminution of the tumor but secondary deposits are common. Amputation of the affected limb following a course of irradiation is generally preferred. This should be done through the bone proximal to the affected bone, if possible. Resection of certain bones, e g, fibula, ulna or scapula is possible if the tumor is present. The metastases of Ewing's tumors are also radiosensitive and are frequently found in other bones (skull, sternum, vertebrae). Pulmonary metastases are common.

Multiple Myeloma (Plasma Cell Myeloma)

This tumor is rare and occurs chiefly in adults over forty-five years of age. It is twice as common in males. Its onset is usually insidious; gradually the pain becomes more severe and pathological fracture is common. The tumor develops in the bone marrow, involving the skull, ribs, spine, and pelvis, and long bones in multiple locations.



Fig. 435.—Secondary carcinoma involving the upper shaft of femur, a metastasis from carcinoma of the breast.

Histology.—The cells are round or oval in shape with an eccentrically placed nucleus, arranged diffusely with no intercellular substance. The cell of origin is doubtful, but it is best considered a bone marrow tumors in which the plasma cell (plasmacyte) predominates in frequency.

X-ray Features.—There is bone destruction without compensatory new bone formation. The bone trabeculae and cortex are

destroyed and appear as multiple, clear, punched-out areas. Bence Jones protein is found in the urine in approximately 60% of the cases. The diagnosis may be made by biopsy of the actual bone lesion or in biopsy of the bone marrow (sternal biopsy).

Treatment.—X-ray therapy is used in an effort to alleviate pain. Pathological fractures may heal with the aid of radiotherapy.

Secondary Carcinoma

Metastatic deposits of carcinoma occur in bone from primary carcinoma in any tissue but are chiefly from carcinoma of the breast, prostate, thyroid, kidney, and bronchus. The spine, humerus and femur are most frequently involved. Pathological fractures are common in the metastases from the breast,

thyroid, and kidneys, but rare in those from the prostate.

X-ray Features.—The bone appears mottled due to the localized destruction. There is no periosteal reaction.

Treatment.—The prognosis is hopeless, but pain can be relieved and prolongation of life secured by irradiation. Pathological fractures are to be immobilized for the relief of pain and the fracture sometimes unites.

REFERENCES

- Coley, B. L.: *Neoplasms of Bone and Related Conditions*, New York, 1949, Paul H. Hoeber, Inc.
Geschickter, C. F., and Copeland, M. M.: *Tumors of Bone*, ed. 3, Philadelphia, 1949, J. B. Lippincott Company.
Jaffe, H. L.: "Osteoid-Osteoma", a Benign Osteoblastic Tumor Composed of Osteoid and Atypical Bone, *Arch. Surg.* 31: 709-728, 1935.

CHAPTER XLII

DISORDERS OF THE VERTEBRAL COLUMN

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ANATOMICAL CONSIDERATIONS

The vertebral column consists of 33 vertebrae; they are named in regions. There are 7 cervical, 12 thoracic and 5 lumbar vertebrae which are movable. There are 5 sacral, fused in the adult to form the sacrum, and 4 coccygeal fused to form the coccyx. The total number of vertebrae is almost constant but regional variations frequently occur. As one would expect, we often see transitional vertebrae which have some of the characteristics of two regions. Thoracic vertebrae show most of the common characteristics, but each region has its special form. The 1st and 2nd cervical are entirely individual. The usual parts of a vertebra are the body, the pedicles, the superior and inferior vertebral notches, the laminae, the vertebral arch, the vertebral foramen which is bounded by the vertebral arch and the back of the body, the spinous process, and the transverse processes, the superior and inferior articular processes which project upward and downward from the junction of the pedicle and lamina and have smooth surfaces for articulation with the contiguous vertebrae. In the vertebral column the vertebral foramina form the vertebral canal. The vertebral notches with the articular processes, bodies and discs, form a series of intervertebral foramina for the emergence of spinal nerves and vessels.

Cervical Vertebrae

Cervical vertebrae are small in size and show three foramina—the large vertebral foramen in the middle and small ones in each transverse process. The pedicles are short, the laminae are long, the spinous process is short and bifid and the transverse process enclosing a foramen ends in an anterior and

posterior tubercle. The first cervical vertebra, or *atlas*, has no body and no spinous process. It is formed by a pair of lateral masses united by an anterior and a posterior arch. There is an anterior tubercle for the attachment of ligaments and the transverse processes are large for the attachment of the muscles that rotate the head. The second cervical vertebra, or *axis*, is characterized by the odontoid process which articulates with the anterior arch of the atlas. The spinous process is strong and broad and bifid, whereas the transverse process is small and not bifid.

Thoracic Vertebrae

Thoracic vertebrae have facets on the bodies for articulation with the heads of ribs and most have facets on the transverse processes for the tubercles of ribs. The bodies are large, the laminae are broad, and each overlaps the lamina below, the vertebral foramen is circular, and the spine is long and slender, and the transverse processes are thick.

Lumbar Vertebrae

Lumbar vertebrae show a large body, pedicles are short and strong, the laminae are thick, and the vertebral foramen is large. The spinous process is almost horizontal, and the transverse processes are long and slender. The articular processes are large, the superior ones being concave, facing medially and the inferior are convex, facing laterally.

Sacral Vertebrae

The sacral vertebrae, five in number, are fused to form the sacrum, which is triangular in shape. The base is directed upward and forward and its projecting anterior part

destroyed and appear as multiple, clear, punched-out areas. Bence Jones protein is found in the urine in approximately 60% of the cases. The diagnosis may be made by biopsy of the actual bone lesion or in biopsy of the bone marrow (sternal biopsy).

Treatment.—X-ray therapy is used in an effort to alleviate pain. Pathological fractures may heal with the aid of radiotherapy.

Secondary Carcinoma

Metastatic deposits of carcinoma occur in bone from primary carcinoma in any tissue but are chiefly from carcinoma of the breast, prostate, thyroid, kidney, and bronchus. The spine, humerus and femur are most frequently involved. Pathological fractures are common in the metastases from the breast,

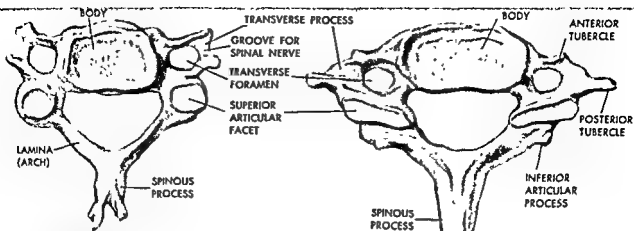
thyroid, and kidneys, but rare in those from the prostate.

X-ray Features.—The bone appears mottled due to the localized destruction. There is no periosteal reaction.

Treatment.—The prognosis is hopeless, but pain can be relieved and prolongation of life secured by irradiation. Pathological fractures are to be immobilized for the relief of pain and the fracture sometimes unites.

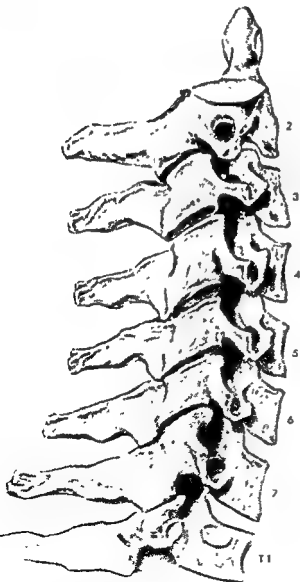
REFERENCES

- Coley, M. L.: *Neoplasms of Bone and Related Conditions*, New York, 1949, Paul B. Hoeber, Inc.
 Geschickter, C. F., and Copeland, M. M.: *Tumors of Bone*, ed. 3, Philadelphia, 1949, J. B. Lippincott Company.
 Jaffe, H. L.: "Osteoid-Osteoma", a Benign Osteoblastic Tumor Composed of Osteoid and Atypical Bone, *Arch. Surg.* 31: 709-728, 1935.

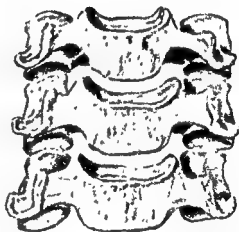


4TH CERVICAL VERTEBRA
VIEWED FROM ABOVE

7th CERVICAL VERTEBRA
(VIEWED FROM ABOVE)



THE 2ND TO 7TH CERVICAL VERTEBRAE
VIEWED FROM THE RIGHT SIDE



THE 3RD, 4TH and 5TH
CERVICAL VERTEBRAE
VIEWED FROM IN FRONT

is called the promontory. Behind the fused bodies the vertebral foramen leads into the sacral canal. The fused lateral masses form the ala of the sacrum, spreading laterally from the body. The apex is formed by the small lower surface of the body of the fifth sacral vertebra.

Coccygeal Vertebrae

The coccygeal vertebrae are variable from three to five and fused to form the coccyx, which is triangular in shape, with its base proximal and its apex distal. It consists of the rudimentary bodies of the vertebrae, more often fused than not but commonly irregular in shape.

Ossification of Vertebrae

The typical vertebra has three primary centers. One appears in the bodies of the lower thoracic region at about the tenth week and these spread up and down, appearing in all bodies by the 20th week, except the lower sacral ones which appear at the 30th week and the coccygeal ones which appear after birth. Two appear at the bases of the superior articular processes in the upper cervical region about the 7th week and extend downward, reaching the sacrum about the 20th week. Ossification extends from these two centers into the vertebral arch, processes, and lateral parts of the body. The central and lateral parts of the body begin to unite about the 3rd year in the neck and the process is complete in all regions by the 7th year. Lamellar ossification is completed after birth, first in the lumbar region, extending upward and downward becoming complete in the cervical at the 15th month and in the sacral about the 10th year. After union of the laminae, ossification extends into the spinous processes.

Five secondary centers appear at puberty and unite with the rest of the bones by the 25th year. There is one for the tip of the spinous process, one at the end of each transverse process, and an annular one on the upper and lower surface of the body.

Joints of Vertebral Column

The bodies of movable vertebrae are united by fibrocartilaginous discs, aided by longitudinal ligaments. In the cervical region, there are also capsules of small synovial joints between their lateral parts. The articular processes are united by capsules of synovial joints. The vertebral arches are united by the intertransverse, interspinous, supraspinous ligaments and ligamenta flava. The intervertebral discs are attached firmly to the articular cartilage which is seen on the contiguous surfaces of the bodies. The disc forms a dense annulus fibrosus in the periphery, and softens toward the center, forming the nucleus pulposus. In the cervical region they do not reach the posterolateral edges of the bodies, due to the presence of small synovial joints. The anterior longitudinal ligament is thick and strong, extending from the anterior arch of the atlas to the sacrum, attaching to the fronts of bodies and to the intervertebral discs. The posterior longitudinal ligament lies in the anterior wall of the vertebral canal, from the axis to the sacrum, attaching to the backs of the bodies and to the intervertebral discs. The ligamenta flava are strong elastic sheets that connect the laminae of contiguous vertebrae, laterally blending with the synovial joint capsule. The interspinous ligaments are attached to adjacent spines from base to tip and the supraspinous ligaments form a long continuous band connecting the tips of the spinous processes along the whole length of the vertebral column. This is thickened in the neck to form the ligamentum nuchae, of importance because of its occipital and muscular connections.

The Vertebral Column as a Whole

The vertebral column may be considered as a central post for support of the body with a complex arrangement of its component parts, supported by strong ligaments and powerful muscles. There are four normal curves, two with their convexities for-

ward in the cervical and lumbar regions and two with their convexities backward in the thoracic and sacral regions. These are smooth curves with a moderate variability within the range of normal, beyond which they are considered pathological; named *lordosis* and *kyphosis*, respectively. The posterior aspect of the vertebral column should present a straight vertical line, or just a trace of right thoracic curve being considered within the normal limit. Any appreciable curve is considered pathological, named *scoliosis*, and designated right or left with its region, according to the side of the convexity. There are, of course, variations with age. The child does not assume the normal curves until he is standing and walking. In old age there is a tendency for the spine to assume a smooth posterior curve throughout. Movements of the spine are carried out by a complex interaction of the enveloping musculature. Flexibility is provided by the compressibility of the intervertebral discs and the gliding action of the synovial joints. The greatest stability is in the erect posture, by virtue of the interlocking articular processes. In the cervical region anteroposterior movement is freely carried out. Lateral flexion is only possible by a combination of torsion and rotation of the vertebrae. In the thoracic region anteroposterior movement is greatly limited by the vertical position of the articular processes. Lateral flexion is much freer and there is quite a good range of rotation. In the lumbar region flexion is very free, whereas true rotation is negligible. The apparent movement is a combination of flexion and circumduction. Alterations from the normal range of appearance and function range from postural defects to traumatic and diseased processes.

DEVELOPMENTAL OR CONGENITAL DEFECTS

The most frequent congenital anomaly of the vertebral column is *spina bifida*. (See Neurosurgical Section.) Radiological studies have shown that about 30% of all spines

show some variation from the normal due to faulty development. Some of these anomalies are incompatible with life and others are of little or no consequence.

Hemivertebrae

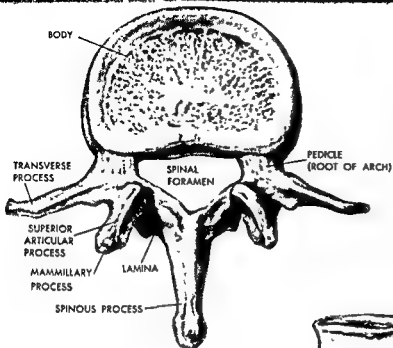
The segments of cartilaginous vertebrae in the embryo may fail to unite and two centers of ossification appear instead of one. Different rates of growth in the two segments may give rise to a congenital scoliosis; persistence of the separation between the two segments may be followed by an anterior *spina bifida*. Development in one segment only produces a true hemivertebra, resulting in a sharp lateral curvature of the spine.

Synostosis

If two adjacent vertebral bodies fuse during their development a synostosis or congenital block vertebra results. The Klippel-Feil syndrome is a synostosis of lower cervical vertebrae, associated with *spina bifida* producing a short and rigid neck. Occipitalization is a synostosis of the atlas with the occiput. Sacralization is a fusion of the 5th lumbar with the sacrum.

Spina Bifida Occulta

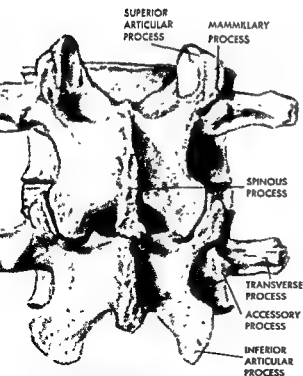
Spina bifida occulta as opposed to the true *spina bifida* shows little or no neurological involvement. There is usually a defect in the neural arch of several vertebrae, the commonest location being in the lumbosacral region. Brailsford gives an incidence of 6% in the 5th lumbar and of 11% in the first two sacral segments. These defects are commonly stated to be of no consequence, but approximately 20% of them do show some *pes cavus* deformity or other neuromuscular anomaly of the lower extremities. Often there is limitation of straight leg raising due to shortening in the hamstring muscles, and often there is an equinus of the foot or at least a limitation of dorsiflexion, due to shortening of the calf muscles.



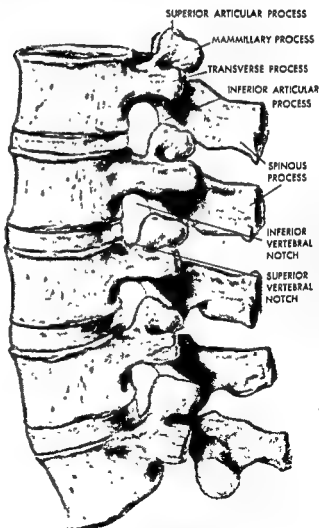
2ND LUMBAR VERTEBRA
VIEWED FROM ABOVE



INTERVERTEBRAL DISC



3RD and 4TH LUMBAR
VERTEBRAE VIEWED
FROM BEHIND



THE LUMBAR VERTEBRAE
VIEWED FROM THE LEFT SIDE

H. H. H. H.



FIG. 437 —Synostosis of lower cervical vertebrae.

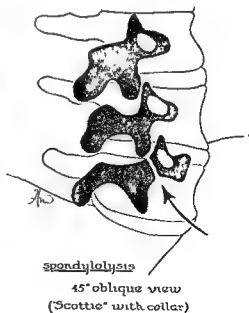


Fig. 438 —Spondylolysis showing defect in pars interarticularis.

Spondylolysis

This is a defect in the neural arch, occurring between the facet articulations. With this defect in the pars interarticularis there is only soft tissue stabilization at this level of the spine. Displacement is accompanied by soft tissue separation at the defect, as well as at the intervertebral disc, and asso-

involve the 4th lumbar, and they are rarely seen elsewhere. In some the displacement occurs soon after birth. Freiberg believes that most of the displacements occur about 12 years of age. Undoubtedly, many do not occur until there is some unusual stress on this segment of the spine. This might follow heavy weight lifting, or some illness with



Fig 436—Multiple congenital anomalies showing hemivertebra at L1.

ciated ligaments. It may produce a traction lesion by the nerve roots being dragged against the vertebral notch. This displacement is known as spondylolisthesis and is commonest in the 5th lumbar vertebra.

Spondylolisthesis

In spondylolisthesis 90% of the defects occur at the 5th lumbar, most of the others

muscular impairment, or obesity with loss of muscular power after giving up athletic activities. Adaptive changes in the shape of the vertebral body would serve to indicate that the displacement had occurred during the growth period. Both spondylolysis and spondylolisthesis commonly give rise to the symptoms that we associate with ligamentous strain. It is not unusual for them to be en-

There was no evidence of neurological involvement; in fact, the patient refused treatment once his curiosity was satisfied as to why his ribs were resting on the pelvis. It is usual, however, for the displacement to be less than a complete dislocation. Traumatic spondylolisthesis, as a major hyperextension injury producing a fracture-dislocation along the same path as the usual congenital defect, is a rare occurrence.

Clinical Features.—The majority of patients do not complain of symptoms until well into adult life. It is often difficult to determine just when the displacement did occur. Usually, they give a history of some fairly recent injury to which they attribute their trouble. They are often incredulous when told that it is congenital in origin. Pain is not constant but is associated with certain movements, particularly stooping and lifting. Usually there are no neurological changes, but occasionally a typical sciatic syndrome is present. When the displacement is appreciable, there is a sharp depression above the 5th lumbar spinous process which is increased with hyperextension. The lumbar region may appear shortened, and the sacral region appear lengthened. If there is marked displacement in a pregnant woman, it may constitute an obstruction to normal delivery. The condition is most clearly seen in oblique x-rays, although if there is significant displacement, it is well seen in the lateral view.

Treatment.—Many of these patients are free of symptoms, due to the natural compensation of powerful musculature. It is not surprising, therefore, that many of those who do have symptoms, are relieved by exercises which develop the erector spinae. Strain also is reduced by development of the hip flexors, enabling them to diminish the pelvic inclination, and flatten the lumbar lordosis. These measures may be aided by the use of a lumbosacral corset or brace, which reduces the painful movements. If pain is severe, or if freedom from restrictive measures is desired, or if there is in-

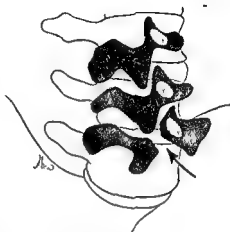
creasing displacement, spinal fusion is indicated. Reduction of the displacement may be obtained when it has occurred recently, by the method described by Watson-Jones. This consists of vertical traction on the flexed hips with the shoulders resting on a table, so that the pelvis is drawn forward against the body weight, if necessary supplemented by the downward traction of a sling around the abdomen. If correction is obtained, a plaster spica is applied and posterior spinal fusion carried out through a window in the cast. In the presence of a rigid deformity, operative reduction is a formidable procedure, and is not worth while. Spinal fusion should be carried out in the position of deformity. Anterior fusion of the 5th lumbar body to the sacrum by an abdominal approach, appears mechanically preferable, but the mortality rate has been excessive. Posterior spinal fusion is considered the method of choice. The area of fusion is subjected to a good deal of shear. Therefore, the method used should produce a massive area of fusion, and it should be protected until it is mature. This reduces the very real risk of pseudarthrosis. The complete Hibbs procedure, with excision of the interarticular joints, and the loose vertebral arch, supplemented by a massive "H" shaped iliac graft, seems to fulfill these requirements.

Kissing Spines

This is a developmental variation; in which there is contact between deformed spinous processes, giving rise to pain, and usually limitation of extension of the spine. An x-ray of the spine which gives good detail in the vertebral body region, does not show the spinous processes so well, and requires close inspection. There may be a false joint or condensation of bone at the points of contact. A similar condition is seen as a complication of spinal fusion. If the surgeon fails to preserve the soft tissue between the spinous processes at the limits of his fusion, then the graft or subsequent

tirely symptomless by virtue of adaptive changes in the ligaments, and compensation by powerful muscle control. Most of the displacements are rigid in their position of deformity, but occasionally a good deal of

instability can be demonstrated, and the patient may be conscious of slipping, occurring during certain movements. The most extreme spondylolisthesis seen showed lumbar 3, 4 and 5 lying in front of the sacrum.



spondylolisthesis

45° oblique view
("Scottie" decapitated)

Fig 439—Spondylolisthesis showing displacement at the pars interarticularis



Fig 440—Spondylolisthesis showing forward displacement at L5 to S1.

more vulnerable, and may be the lesion requiring treatment. Thorough investigation is essential.

Treatment.—

1. *Conservative.*—

(a) Bed rest is indicated at the start of an acute exacerbation of symptoms.

(b) Limitation of painful movement is achieved with a Harris brace or a plaster corset.

(c) Novocain infiltration is often of diagnostic and therapeutic value.

(d) Deep heat, massage, and exercises are used as the acute phase subsides.

Cervical Rib and Scalenus Anticus Syndrome

Vertebrae which do not normally have ribs occasionally develop rudimentary ones. This is most commonly seen in the 7th cervical vertebra. The cervical rib often gives rise to symptoms either directly or by its ligaments, or attached scalene muscles, producing traction on parts of the brachial plexus. The cervical rib may compress the subclavian artery or the cords of the brachial plexus against the posterior border of the scalenus anticus muscle. The vein lies in front of this muscle and escapes. If symptoms are due to traction, they are aggravated by movements which drag on these



Fig 442.—Congenital torticollis

(e) The maintenance of powerful musculature by daily remedial exercises is designed to prevent recurrences.

2. *Operative.*—Indicated if adequate conservative treatment fails or if heavy use of the back is necessary.

(a) *Transversectomy* is indicated if one is certain this anomaly accounts for the symptom-complex. It allows early mobilization of the spine with a short convalescence.

(b) *Spinal Fusion* is often preferable as it strengthens the spine. It will also cure the symptoms, of almost any low back disorder, associated with this condition

two structures. If due to pressure, they may be constant. Many cervical ribs give no trouble. Others threaten gangrene or permanent paralysis.

Treatment.—Removal of the cervical rib is the only satisfactory means of relief. It requires a careful technique.

Congenital Torticollis

There are many causes for *wry neck*—hysterical, habit, ocular, bony, muscular and nerve lesions. It may be associated with congenital, paralytic, traumatic or inflammatory changes.

callus may impinge on the adjacent spinous process with spinal movements, and give rise to pain of the same type. A cure of both conditions is simply to excise the offending bony protuberance, and suture viable soft tissue between them.

Sacralized Transverse Process of the 5th Lumbar Vertebra

This is a congenital variation from the normal, in which there is enlargement of one or both transverse processes. It, or they, articulate with the sacrum, or sacrum

opposite side. This would produce recurrent ligamentous injury. The fourth lumbar nerve passes over the prominent transverse process. This with spinal movement may give rise to a traction lesion. If the enlarged transverse process is just short of articulating with the ilium, it may, with spinal movement, give rise to intermittent impingement. This would nip the intervening soft tissue and give rise to traumatic inflammatory changes. It could also produce an adventitious bursa, with recurrent bursitis.



Fig. 441 —Sacralized transverse process of 5th lumbar vertebra

and ilium. The term transitional vertebra is used to indicate that it has some characteristics of both lumbar and sacral segments. Often the articulation is structurally poor, and suffers traumatic or degenerative change readily. This will produce pain on the same side. Commonly, however, pain is on the opposite side. This is probably due to strain on the weaker side. With movement to the side of the bony buttress there may be leverage against the ligaments on the

Cases have been described in which osteophytes from an arthritis in the false joint have encroached on the intervertebral foramen, affecting the emerging nerves and vessels. The counterpart of this syndrome occurs with lumbarization of the first sacral segment. One must recognize that this anomaly may be present, and symptoms be due to one of the other numerous causes of low back pain. For instance, the intervertebral disc immediately above is rendered

placed centrally at these levels and an attached protractor with a plumb line to give the reading. The inclination of the pelvis is increased by contraction of the hip flexors and decreased by the hip extensors, any alteration affecting the entire posture of the spine.

Lumbar Lordosis

Lumbar lordosis is a deviation from normal posture, in which the pelvis is tilted forward. In order to maintain the line of weight-bearing, it is necessary to have an increased concavity in the lumbar region. There is perhaps a little increase in the thoracic kyphosis, but this is not a prominent feature. The whole appearance is altered by correcting the pelvic tilt.

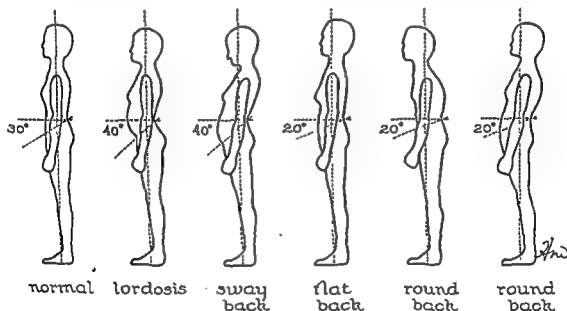


Fig 443 —Types of postural defects.

Sway Back

Sway back is a combination of forward tilting of the pelvis and a dorsolumbar kyphosis. So that, instead of correcting the line of weight-bearing by increasing the lumbar concavity, the entire spine is bent backward, rather markedly at the lumbosacral level. The pelvis projects forward, with the body and the legs sloping backward.

Round Back

Round back is characterized by a decreased pelvic inclination in combination with a dorsolumbar kyphosis. With this, the line of gravity may be corrected by the trunk being held forward, producing obliteration of the lumbar curve. It may also be corrected by bringing the pelvis forward, and angulating the trunk backward at the lumbosacral level, producing an appearance somewhat similar to the sway back posture.

Flat Back

The flat back is produced by the pelvic inclination being reduced and is balanced by a reduction in the lumbar curve. This type of variation from the normal is less

noticeable than the others, but it does give rise to trouble in later life, and should be corrected

Round Shoulders

This is a mixed type of postural defect in which the scapular muscles sag, allowing the shoulders to fall downward and forward. It is further aggravated by some increase in the thoracic curve, and forward inclination of the head and neck.

The congenital variety is due to a retardation in lengthening of the sternomastoid muscle. The disturbance is probably due to interference with the arterial blood supply—analogue to a Volkmann contracture.

Clinical Picture.—Soon after birth, a firm lump may be felt in the sternomastoid. This disappears spontaneously, but an observant mother may notice that there is some restriction of neck movements. Gradually a deformity develops due to the related shortening of the muscle. There is a flexion of the head to the side of the lesion and a rotation of the face to the opposite side. If the condition is not treated, asymmetry of the face develops. There is ocular adjustment to the position of deformity. This becomes a problem in late corrections. There may be secondary structural changes, giving rise to an upper thoracic scoliosis and elevation of the shoulder. These changes constitute a very ugly deformity. Double torticollis and posterior torticollis are rare. The latter is due to similar changes in the trapezius and other posterior muscles.

Treatment.—It appears that some infants develop the lump in the sternomastoid muscle and go on to complete and spontaneous recovery. If, however, any of the other signs develop, it is imperative to commence early treatment.

1 *Conservative treatment* consists of passive stretchings. The mother is trained in the technique and carries it out three times daily. Clinical review is essential at frequent intervals. It should be discontinued unless effective.

2. *Operative treatment* is indicated with the appearance of any appreciable deformity. The sooner it is done the better. It consists of an open division of the sternal and clavicular heads of the sternomastoid muscle. Postoperative splintage in the overcorrected position is used till soft tissue healing is consolidated. This is followed by postural exercises and passive stretchings. Review at

suitable intervals up to adolescence is advisable. Any recurrence should be prevented or corrected before it develops into a real disability.

Postural Defects

Postural defects are those which cannot be attributed to any traumatic or disease process and which can be corrected by voluntary efforts. They are often associated with postural defects elsewhere in the body. Form and function are interrelated, so that during the growth period a postural defect may be converted into a structural one. Also conversely, during the growth period, minor structural defects may be corrected by the development of a good posture. While in the adult the plasticity of bone is not entirely lost, these changes become negligible. The natural tendency, nevertheless, to compensate for a structural defect can be enhanced by the development of powerful musculature. The maintenance of a poor posture is probably a function of the central nervous system. A habit produces a conditioned reflex, which maintains the altered muscle length as if it were normal. It is common to have weak muscles associated with a faulty posture, but it is not necessarily so. The conscious effort to maintain a correct posture until it becomes a conditioned reflex is probably the most important feature in the treatment. It is apparent that any alteration in the weight-bearing joints may affect spinal posture. It may be a valgus foot with external rotation of the limb and tilting of the pelvis and therefore of the spine; or it may be primarily in the hip, pelvis, or the spinal column. There are various methods of measuring pelvic inclination, but the range of normal variation is wide enough to depend on careful observation. An instrument called a *pelvic inclinometer* permits a reading of pelvic inclination from the upper part of the symphysis pubis to the level of the posterior superior spines of the ilium. The instrument has two blades to be

and muscular rigidity, and it is well to bear in mind the false localization and reflex muscular effects which may occur from the involvement of deep-lying somatic structures and also of certain viscera. Sciatica, for instance, may arise reflexly from a gluteal muscle lesion, from a lumbosacral ligamentous lesion, or osteoarthritic changes in the facet articulations, as well as from direct pressure on the nerve root by protrusion of an intervertebral disc.

Myofascial Injuries

These injuries may produce a sciatic scoliosis with lumbosacral tenderness, occasionally tenderness along the course of the sciatic nerve, limitation of spinal movement (most marked limitation being of flexion) and limitation of straight leg raising. The attachments of the injured muscles are particularly tender, and there may be moderate tenderness over all of the muscle groups. The symptoms are relieved by heat and rest and are aggravated by activity. Novocain infiltration of the points of maximum tenderness is useful both as a test and for relief of symptoms. It is considered confirmatory when insertion of the needle aggravates both the local and referred pain, and the injection of the local anesthetic relieves both of these, and is followed by the restoration of straight leg raising. Occasionally, one sees systemic reactions, characterized by giddiness, rapid pulse, sweating and a drop in blood pressure. If severe, these symptoms may be relieved by the injection of 1 c.c. of 1/1000 Adrenalin. The antihistamine drugs are ineffective. Also, there may be a delayed painful reaction as the effect of the local anesthetic disappears, so that for a while the pain is far worse than the original one. This may be relieved by the use of analgesics. Treatment should have as its object the healing of damaged tissues with the prevention of extensive adhesions. Therefore, in the acute phase, rest and support of the part to relieve pain are

indicated. Depending on the severity of symptoms, this may be achieved by strapping, a tight corset, a brace, or plaster jacket. Bed rest in the early stages is often advisable. Radiant heat and light massage are soothing in their effects. Short wave diathermy and heavy massage will aggravate the condition, although they are often useful with active exercises as the acute phase subsides.

Chronic Strain or improperly treated acute injuries may result in myofascial adhesions. They give rise to recurrent low back pain and sciatica, often with acute exacerbations. In some of these there is contracture of the gluteal fascia below the posterior superior spine. Roberts and Heyman have advised stripping of the posterior superior spine. In others, the contracture is more anterior in the tensor fascia lata and the iliotibial band. This may be demonstrated by the Ober test, which consists of having the patient lie on his good side and flexing the hips to flatten the lumbar spine. The affected leg is flexed to a right angle at the knee while the hip is steadied and then is abducted and extended, bringing the thigh in line with the trunk. The abduction contracture of the iliotibial band can then be felt as it maintains the limb in the abducted position. Ober has recommended division of the iliotibial band and fascia lata with the intermuscular septa for relief of this condition. When the original symptoms were due to involvement of the erector spinae muscles and there is no abduction contracture of the hip, manipulation of the spine is often recommended. Due, however, to the danger of displacing an intervertebral disc, this is considered a dangerous procedure. It also appears that in the vast majority of these conditions, an equally satisfactory result can be obtained by conservative measures. These consist of Novocain infiltrations, massage, short wave diathermy, and spinal exercises, sometimes supplemented by the use of a sup-

Postural Scoliosis

This is a functional lateral curvature which can be fully corrected by voluntary effort. It is in the same category as other postural defects, and does not give rise to serious structural changes. There is usually a smooth total curve of the lumbar and dorsal regions, with little or no rotation of the vertebrae and no secondary changes in



Fig. 444—Fatigue posture (sway back)

the thoracic cage. The curve disappears when the spine is flexed. It sometimes disappears when sitting and can be corrected by muscular efforts when standing. It is often intermittent and associated with fatigue. It is to be distinguished from the lateral curvature which occurs with a short

extremity. Variations in leg length up to a quarter of an inch are of no consequence and do not require correction. Children who show a postural scoliosis will usually correct it spontaneously with growth and muscular development. It is, however, wise to check up on the child's general health and to teach him postural exercises.

Treatment of Postural Defects.—Once the mechanism of a postural defect is understood, then the exercises necessary to correct it are easily determined. Advantage is taken of the reciprocal innervation of antagonistic muscle groups. If an extensor group of muscles is contracted in carrying out a movement, the flexor group relaxes sufficiently to permit the movement, but contracts sufficiently to control the joint. If the extension is against resistance, control of the joint is obtained between these two forces, and the reflex inhibition of the flexors is much more profound. If, therefore, we wish to correct the increased pelvic inclination of a lumbar lordosis or a sway back defect, we should develop the gluteal muscles and relax the hip flexors. Gluteal resistance exercises are, therefore, prescribed. If an exercise is carried out from a correct postural position, and returns to this position, and is repeated frequently enough, the normal postural reflex is re-established. It is not sufficient to simply build up muscular power by general exercises. It is important then, to convert the new posture into all the daily activities, until it is finally maintained without conscious effort.

TRAUMATIC DISORDERS OF THE VERTEBRAL COLUMN

The complex arrangement of the vertebral column is susceptible to a wide range of injuries: muscular, ligamentous, discoid, cartilaginous and bony, as well as neurological. Pain arising in any of these structures may be referred to the skin distribution of the segmental innervation of the part concerned. It is accompanied by tenderness

Cervical Discs

If the protrusion is central and large enough, it produces a cord lesion resembling a cord tumor. Operative removal is imperative in order to prevent permanent damage. Fortunately these midline massive extrusions of the nucleus are rare.

Commonly the protrusion is lateral, and involvement of the nerve root is variable and usually reversible.

Clinical Features.—Some history of trauma, recent or remote, can usually be established. One would expect extrusion of the nucleus if symptoms were persistent from the time of injury. The vast majority of cases show a considerable time interval between the injury and the onset of symptoms. There is a predilection to the middle age group, so that degeneration undoubtedly plays an important part. It is a traumatic degenerative change in a disc which has probably lost some of its resilience. The fluid content of an intervertebral disc decreases with age, and this dehydration is appreciable at about 40 years of age.

Limitation of movement due to pain is seen early. There is referral of pain according to the segment involved. Jarring of the spine in any way is apt to aggravate symptoms. Maneuvers which increase cerebrospinal fluid pressure produce a sharp stab of pain, e.g., coughing, sneezing, and straining. There are troublesome subjective symptoms of numbness, pain, and coldness, before sensory involvement can be demonstrated. Muscular weakness may be detected by comparison of power with the unaffected limb. Tenderness of the spinous process at the involved level helps localization. The affected dermatome can be mapped out if there is objective sensory change.

A disc protrusion C5 to 6 is likely to produce sensory changes in the thumb and index finger. There may be loss in power of elbow flexion. The biceps jerk is diminished or absent.

If the involvement is at C6 to 7, the middle and ring fingers may show paresthesia. There will be loss of power of extension in the elbow, and the triceps reflex is diminished or absent.

X-ray examination may be normal in a recent involvement. When the condition has been present long enough for adaptive changes to occur, we expect to see narrowing of the intervertebral space. Often if films are taken in flexion and extension, an alteration in the curve can be detected. There is loss of mobility at the level of a disc lesion. Further progression will result in peripheral osteophytosis, and if this projects posteriorly, it is direct evidence of encroachment on the vertebral canal.

Treatment.—

1. *Conservative*.—It is generally recognized that a cervical disc protrusion responds well to conservative measures. These are:

(1) Short wave diathermy and massage to relieve the secondary muscle spasm and pain.

(2) Analgesics and sedatives for the same reason.

(3) Head traction to reduce the herniation of the annulus; this may be by a small weight for a long period or a larger weight for short periods. A usual prescription is 20 pounds for 20 minutes, three times weekly for 3 or 4 weeks. Occasionally a 5 pound weight is used almost continuously for a week, in bed, and succeeds where the first has failed. A well-fitted head halter with ropes, pulleys, and weights is used.

(4) Cervical splintage may be useful to limit painful movement, and there is a variety of methods: (a) Plaster "doll's collar." (b) Molded leather collar. (c) Plastic mold collar. (d) High felt collar, etc.

These measures should clear up symptoms in 90% of cases. Permanent relief is obtained by developing powerful cervical musculature and avoiding trauma to the region.

Lumbosacral and Sacroiliac Strains

The ligaments supporting the lumbosacral and sacroiliac articulations are so powerful that it must be indeed rare to have an actual tearing of these structures. Ligaments, however, do not stand up well to continuous strain, and with muscular insufficiency and postural defects, they frequently give rise to symptoms. There is protective muscle spasm, with pain both local and referred, which may be present in varying degrees according to the severity of the strain. Usually there is relief of symptoms with bed rest, which continues into the morning, but the symptoms recur with the onset of muscular fatigue. A minor injury will often produce an acute exacerbation of the symptoms. Testing the joints while symptoms are present will produce an exacerbation, but often if examined in a period of remission, localization cannot be determined. The mechanism of injury may be of value, flexion injury being likely to affect the lumbosacral articulation, and twisting strains the sacroiliac joint. The latter is very powerful except during pregnancy, when due to the hormone relaxin, the pelvic ligaments are relaxed. The sacroiliac joint may be tested by having the patient lie on his side, and compressing the iliac crests, also lying on the back and forcing down on both anterior superior spines, or lying on the back, hyperextending one hip over the examining table. If positive, each of these tests reproduces pain and tenderness at the sacroiliac level. With the patient lying prone, hyperextension tests both joints, being considered positive according to the localization of the pain. Pain with flexion is often well localized in lumbosacral strains. Rotation tests are not very useful, but if positive, point to the sacroiliac joint. The vast majority of so-called sacroiliac strains are shown by these tests to be lumbosacral ones. Treatment is the same as described for myofascial injuries, except that Novocain infiltration is much less use-

ful, and very occasionally for persistent cases a fusion of the affected joint may be required.

Intervertebral Disc Protrusions

There are two common regions for intervertebral disc protrusion to give rise to symptoms. The lower lumbar L 4 to 5 and L 5 to S 1 account for probably 90% of disc protrusions. The lower cervical C 5 to 6 and C 6 to 7 account for most of the others, the former being twice as frequent. There are many more symptomless disc protrusions than ones that give rise to trouble. Myelograms suggest a proportion of 4 to 1, but anatomical dissections suggest it is very much higher. It is obvious that a "proved disc" is not proof that it causes the symptoms. All other causes for the symptom-complex must be considered in the clinical appraisal. There are many vertebral disorders in which some discoid anomaly is associated. These are described elsewhere in this section—see.

- 1 Spondylolisthesis
- 2 Pott's disease.
- 3 Osteomyelitis of the spine
- 4 Hypertrophic spondylitis
- 5 Ankylosing spondylitis
- 6 Adolescent kyphosis
- 7 Senile kyphosis
- 8 Senile osteoporosis.

The modern glib diagnosis of "disc" for almost every vertebral column disorder has, therefore, a grain of truth in it. It is, however, a retrograde development. The term should be reserved for the clear-cut symptom complex associated with extrusion of the nucleus pulposus or herniation of the annulus fibrosus into the vertebral canal, causing cord or nerve root irritation. The rarer nuclear prolapse or sequestered disc is likely to give rise to intractable nerve pain or cord symptoms and usually requires operative measures. The commoner, herniation of the annulus, is usually reversible, gives rise to intermittent symptoms and responds to a variety of treatments.

extension. If at this point the foot is dorsiflexed, there is further aggravation of the pain.

There is tenderness of the regional spinous processes, often maximal at the level of the lesion.

Hypesthesia or paresthesia should be mapped out and aids localization according to the dermatome pattern.

Muscle weakness is a common finding, particularly extensor hallucis longus in L5 to S1 protrusions. The knee jerk is diminished or absent in L3 to 4 disc lesions, the ankle jerk in L4 to 5 and L5 to S1 protrusions.

Rectal examination is done to exclude intrapelvic neoplasm as a possible cause for sciatica.

X-rays are of considerable value, both for confirmatory and exclusion evidence. There may be no changes in a recent protrusion, but later there is reduction of the interspace, perhaps loss of mobility in bending films, possibly posterior osteophyte encroachment on the vertebral canal. Anteroposterior, lateral, and oblique films, at least, should be taken to help rule out congenital, infectious, and static disorders.

Myelogram is a special type of x-ray study in which 3 c.c. to 9 c.c. of Pantopaque are injected into the vertebral canal by ordinary lumbar puncture. By changing positions of the tilting table on which the patient lies, this can be gravitated as desired to outline the vertebral canal. Any encroachment by a disc protrusion is recognized by a filling defect. If the findings are compatible with the clinical picture, it is considered significant. Symptomless protrusions thus demonstrated are disregarded. The test is restricted to cases in which operative treatment is contemplated.

Treatment.—

1. Conservative.—

(1) Short wave diathermy, massage, and flexion exercises often suffice to clear up mild attacks

(2) Analgesics, sedatives and local anesthetic infiltration of points of maximal tenderness aid the symptomatic relief.

(3) Bed rest may offer a good deal of relief. Fracture boards under the mattress may give some firm support to the part. More often the position of flexion, with pillows under shoulders and knees, gives relief by positional reduction of the tension on the sciatic nerve.

(4) A flexion plaster corset is often effective. Lower lumbar flexion is obtained with the forward tilt of the pelvis when sitting. A cast applied in this position is worn for about 6 weeks. Exercises are started as soon as tolerated.

(5) A Harris brace is useful to control chronic or recurrent attacks by limiting painful movement. It should be fitted with reduced lumbar lordosis.

2 Operative treatment is indicated:

(1) When the symptoms are so severe or persistent that it is thought conservative measures will be ineffective. These are usually sequestered nuclear protrusions or very large protrusions of the annulus.

(2) When adequate conservative treatment has failed to give relief.

(3) When motor, sensory, and reflex changes are severe enough to threaten permanent nerve damage.

(4) When restrictions about the heavy use of the back cannot be accepted.

The localization is confirmed by Pantopaque myelogram.

An interlaminar approach is used and the offending disc is excised. Usually sequestration of the nucleus or adhesions of the annulus fibrosus herniation, as a result of delay in adequate treatment, accounts for the failure of conservative treatment.

Incomplete removal of the intervertebral disc is to be avoided as it could result in a further protrusion and recurrence of the symptoms. It is wise to finish up with a localized spinal fusion, as the long-term results are better. The writer prefers the pos-

2. *Operative* treatment is as described for lumbar discs. The cord must be carefully protected and a careful technique is required.

Lumbar Discs

The involvement of lumbar discs is currently believed to be the commonest cause of low back pain. Certainly there has been a tremendous interest in the subject since the early work of Mixter and Barr in 1934. Prior to that time, the condition was not commonly recognized. It is probably now being diagnosed too frequently on insufficient evidence. The whole problem of low back pain must be understood in order to place the component parts in a proper perspective.

The lumbar discs, at the lower two segments, are subject to a good deal of stress, just as are the other structures at this level. When there is a nucleus pulposus extrusion, symptoms of nerve root irritation are usually severe and persistent. Bulging of the annulus fibrosus, which viewed from the vertebral canal aspect would give a washboard appearance, is often of much less importance. The former condition is not reversible, but the latter is often amenable to simple measures.

Clinical Features.—Symptoms fall into two main categories, those of gradual onset which appear compatible with a degenerative lesion, and those of sudden onset which suggest a traumatic lesion. Obviously, there are many variable factors and combinations of modes of onset. If there is a central protrusion, one finds bilateral involvement of the lower extremities, and the lesion may be at a higher level than expected. As localization clinically is based on the level of nerve root emergence from the vertebral canal, a central protrusion may affect it a little higher up. The more common lateral disc protrusion is more accurately localized by the dermatome involvement. This is one of the many causes of the symptom complex referred to as lumbago or sciatica. It is

probable that some cases with severe subjective complaints of this nature are minimal disc protrusions, but it is a speculative diagnosis which should not be made. Other more inclusive terms are more in keeping with the probabilities and less likely to lead to misinterpretation. If there are motor, reflex, or sensory changes, then the probability of a protrusion is greater.

With lumbago, regardless of its cause, there is spasm of the erector spinae, flattening of the lumbar lordosis, and painful limitation of movement. In sciatica there is a scoliosis due to unequal muscle spasm in the lumbar muscles and flexion of hip and knee on the affected side. Straight leg raising is limited on both sides but much more on the affected side. Lasègue's test is positive. It consists of flexing the knee and bringing the hip to a right angle. Then as the knee is extended, pain is produced short of full

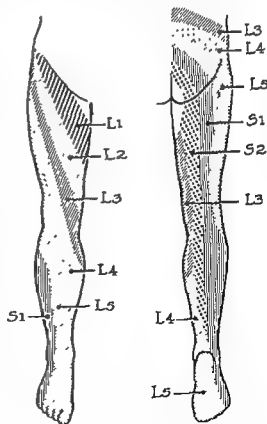


Fig 445—The dermatomes of the leg (after Keegan).

- 4 Dislocations of the cervical spine.
5. Fracture-dislocation without paralysis.
6. Fracture or fracture-dislocation with paralysis.

Fractures of Vertebral Bodies

Etiology.—These injuries constitute the largest number of spinal fractures and are usually of the compression type. The fracture may be in any vertebral body but the majority occur at the cervicodorsal and dorsolumbar junctions. They almost always occur with the spine in flexion, as in a fall from a height. It is quite common to see more than one vertebra involved with this type of injury, and occasionally there may be several normal between two or more compressed bodies.

Diagnosis.—The patient immediately complains of pain. There is muscle spasm of the erector spinae group. Prominence of a spinous process may be noticed, and localized tenderness on palpation of the involved vertebra or vertebrae is of definite significance. The roentgenograms must be as complete as the patient's positioning permits. Stereoscopic views are often valuable, especially where the neural arch is also involved. The lateral view shows irregularity of the vertebral body with a diminution in its vertical measurement. The intervertebral disc space is usually not narrowed. Compression fractures in the dorsolumbar area show more deformity than in other areas.

One must not overlook injuries to other parts of the body when a fracture of the spine has occurred and x-ray studies of these parts should be taken if indicated.

Prognosis.—Healing of a compression fracture of a body is good, and if the deformity has been corrected, any resulting disability can be found in the muscles and ligaments.

Treatment.—One must always rule out a fracture of the pedicle or of an articular facet, in order to exclude the possibility of a fracture with dislocation. Reduction of the fracture by hyperextension is the pro-

cedure to be employed if the body is the only element involved. The technique of reduction can be carried out by one of the following methods: Davis, Rogers, or Watson-Jones. After the fracture has been reduced, a plaster corset is applied from the suprasternal notch to the symphysis pubis. Postreduction films must show perfect reduction of the deformity. Exercises are to be carried out during the immobilization, which is usually between four and six months, depending on the severity of the fracture. The patient is allowed up and about within a few days of immobilization.

Late cases of fracture of the body or those improperly handled may be painful. These should be treated by exercises to develop compensatory curves above and below the old injury, or by bone graft.

A serious complication of fractures of the lumbar vertebrae is retroperitoneal hemorrhage which may cause abdominal ileus.

Fractures of Muscular Processes

(a) Spinous Processes are usually caused by direct violence.

Diagnosis.—Swelling and spasm of muscles occur with local tenderness at the site of injury. X-rays confirm the diagnosis and also indicate whether any other element of the spine is involved.

Treatment.—Rest for a few days, followed by exercises, is usually sufficient. In the cervical area, due to the weight of the head, a Minerva jacket may be necessary.

(b) Transverse Processes occur only in the lumbar area and are caused by muscular violence.

Diagnosis.—There is severe pain especially on lateral bending of the spine, muscle spasm of the lumbar group, and local tenderness about three inches from the midline. Anteroposterior x-rays confirm the diagnosis.

Treatment.—Plaster immobilization with the body in the neutral position is carried out for 4 to 6 weeks. Exercises while in plaster should be done from the beginning

terior spinal fusion by an "H" graft. This maintains the normal posterior relationships of the adjacent vertebrae, making it unnecessary to enlarge the intervertebral foramina. (The nerve root lies in the inferior part of the intervertebral foramen directly between the intervertebral disc anteriorly and the capsule of the apophyseal joint with the

ligamentum flavum posteriorly.) The graft is under physiological compression, which by eliminating shearing strains, encourages bony union. In fact if one could be certain that there was no sequestration of the nucleus pulposus or mature adhesions, the fusion by this method would alone reduce an annulus herniation and effect a cure.

FRACTURES AND DISLOCATIONS OF THE VERTEBRAL COLUMN

J. GORDON PETRIE, M.D.

Emergency Management.—First aid to the person with an injury to the back or neck is of prime importance and may influence the end result. It is now generally recognized that a patient with an injured vertebral column must be moved as a whole without allowing shift of the injured part. The first doctor to examine the patient should carry out an examination of the sensory and motor functions of the extremities; tenderness and deformity of the spinous processes can be quickly detected. If shock is present, it should be treated while other examinations are being done.

The serious cervical injury presents special problems. Temperature must be watched and hyperthermia controlled. Impaired respiratory function makes it important to maintain a free airway. Simple suction may be necessary, and oxygen may lessen cyanosis. Light longitudinal traction with an improvised head halter will provide safety in transportation.

The best method of transporting the victims of thoracic and lumbar spine injuries is on a padded board the size of the ambulance stretcher and securely lashed to the litter. Hypothermia may be present and the patient may require extra covering. Oxygen may help if there has been prevertebral hemorrhage causing impaired heart action. Catheterization and a rectal tube may reduce intra-abdominal pressure.

Serious associated injuries must not be overlooked. With sensory pathways im-

paired the patient may be unaware of other injuries and be unable not only to recognize them, but even to help locate them. Complete notes are made and the time of the examination put down so that any change in the findings may be related to the examination and treatment.

Lumbar puncture may be done in cases showing gross neurological damage. Lack of the normal cardiac and respiratory oscillations and rise of pressure in the manometer on jugular pressure signify that the spinal subarachnoid space is blocked. The inflation of a blood pressure cuff wrapped about the neck and pumped to various pressures provides controlled, reproducible compression that permits differentiation between a partial and a complete block.

The x-ray study is as comprehensive as the patient's positioning permits. All views are taken for stereoscopic study in the anteroposterior, lateral and often oblique positions. X-ray studies of other parts are taken if indicated.

INJURIES TO THE VERTEBRAL COLUMN

Classification of Vertebral Fractures and Dislocations:

1. Fractures of vertebral bodies
2. Fractures of muscular processes—transverse; spinous.
3. Fractures of the neural arch—pedicles, articular facets, laminae.

STATIC DISORDERS OF THE VERTEBRAL COLUMN

A. A. BUTLER, M.D.

Fibrositis

Fibrositis, fibromyositis, and muscular rheumatism are terms used for pain in the soft tissues, characterized by muscle spasm and tenderness, localized at "trigger points" or sites of maximum tenderness, from which pain seems to be referred. Commonly there are associated tender nodules in muscle or subcutaneous tissues which are called "fibrositic nodules."

Etiology.—The cause is unknown; lowered general resistance, exposure to cold, dampness or trauma, fatigue, mental anxiety, metabolic toxins, intestinal intoxication, bacterial toxins, gout, excessive exercise, faulty posture, endocrine disease, muscular disturbances, and so on are variously considered the cause. If we consider it a symptom-complex, rather than a clinical entity, we have a useful term which does not exclude a more accurate diagnosis if it can be established.

Pathology.—Sections of affected tissues show pathological changes of a rather inconsistent nature. Inflammation of interstitial connective tissue of affected muscle, nodular in distribution, is described and is presumed to cause a spastic reaction in surrounding contractile tissue which becomes palpable. The subcutaneous nodules on biopsy appear to show fat herniation through a fibrous capsule, which on cross section is edematous and hemorrhagic.

Clinical Features.—The pain may be acute or insidious in onset. Muscular spasm is usually an outstanding feature. Certain "pain patterns" are recognized as—*torticollis*, affecting sternomastoid, trapezius and cervical muscles—*lumbago*, affecting lumbar and gluteal muscles, and so on. Posture is altered and movements are limited due to the muscle spasm. Passive movements are usually much freer than active ones if carried out slowly, but if abrupt, may precipi-

tate an acute exacerbation. There may be reflex or sensory changes, and straining usually aggravates the pain.

Differential Diagnosis.—The determination of the source of pain is notoriously difficult. We must recognize that the same false localization, and reflex muscular effects, may occur from involvement of deep parietal structures, and certain viscera. Fibrositis of the middle part of the left trapezius may produce an anginal type of pain by virtue of T 2, 3, 4 reflex. Involvement of cervical muscles commonly produces headaches. Fibrositis of dorsolumbar muscles may mimic appendiceal or ovarian pain, T 9 to 12. Scleritis anticus involvement may resemble gall bladder disease, C 3, 4, 5 reflex via the phrenic nerve. Psoas fibrositis may resemble abdominal, genitourinary or vertebral lesions.

These false localizations can be reproduced by the injection of irritating solutions at various levels into paravertebral tissues. At L-1, for instance, pain will radiate to loin, inguinal and scrotal regions with rigidity and deep tenderness in lower abdominal wall, resembling renal colic.

Ischemia is an important factor in producing pain, and there is a great difference in the reaction to exercise, e.g., the intermittent claudication of Buerger's disease and the pain of cardiac ischemia. If a tourniquet is used to produce ischemia in a limb, there is much less pain at rest than with exercise, presumably due to a difference in the chemical changes in the tissues.

Investigation must exclude visceral, neurological, and skeletal disorders to be adequate.

Treatment.—Heat, massage, analgesics, and local injections of Novocain are often successful in clearing up acute attacks. An exercise program and attention to general health are designed to prevent recurrences.

Union of the fracture is a fibrous one and therefore reduction of the fracture is not attempted. Normal activity is encouraged early.

Fractures of the Neural Arch

(a) **Pedicles.** Such fractures, when they occur, are usually in conjunction with those of other elements of the spine

(b) **Articular Facets** are usually involved in cases of fracture-dislocation. It is necessary to have x-rays taken in various projections to demonstrate these features

(c) **Laminae.** Direct violence is usually the cause of these fractures and the fragments may cause direct pressure on the spinal cord.

Treatment.—One must consider whether there is a partial or complete subarachnoid block or whether there is x-ray evidence of the spinal cord being encroached upon by bone, and if there is more than one element of the spine involved by the fracture. If these findings are present, it is felt that early exploration and internal fixation are necessary.

Dislocation of the Cervical Spine

Etiology.—The mechanism of production is one of hyperflexion of the spine. The dislocation may involve one or both facets and may be accompanied by varying degrees of injury to the spinal cord.

Treatment.—Skeletal traction by Crutchfield's or Barton's tongs is the method of choice and has largely displaced the manual manipulative methods. The tongs are placed below the parietal eminences and project into the outer table 3 mm. Following reduction, a Minerva jacket is applied with the head in moderate hyperextension. The jacket is worn for two months after which a plaster or felt collar will give sufficient hyperextension for one additional month. X-rays must be repeated to see that the reduction is being maintained, and if it is not, a fusion operation will be necessary.

Fracture-Dislocation of the Spinal Column Without Neurological Involvement

Etiology.—These injuries are usually caused by severe flexion or extension of the spine. The commonest findings are a compression fracture of a vertebral body with a fracture of one or both articular facets and a dislocation forward of the involved body upon the ones above and below. The pedicles, laminae and spinous processes may be included in the fracture

Nonsurgical Treatment.—For the cervical fracture-dislocations, skeletal traction applied with the Crutchfield or Barton-Cone skull tongs is the best method. Halter traction is satisfactory only in the emergency management. Repeated x-rays are essential to follow the course of reduction. Manometric studies are to be carried out to see whether the traction is satisfactory when a subarachnoid block has been present.

For the thoracic and lumbar fracture-dislocations, traction on the shoulders and lower extremities can be tried with special apparatus. Manometric study should be repeated if a block has been previously demonstrated

Surgical Treatment.—Indications—when spinal block is partial or complete. When the fracture involves more than one element of a vertebra, stabilization is indicated

Exploration is carried out as soon as the condition of the patient permits. Traction is applied when the involved area has been exposed and the spinal cord is under direct inspection. When restoration of the vertebral canal is accomplished, internal fixation is secured by means of bone grafts

Postoperative Management is simplified due to the operation. A Minerva plaster jacket is used for cervical cases, and bilateral casts for injuries lower in the spine.

When bladder control is lost, Munro's tidal irrigation is started at once. If urinary infection cannot be controlled by drainage, suprapubic cystostomy may be necessary

Pressure sores can best be prevented by frequent turning; this is much safer after internal fixation of the spine

up, there is a good deal of improvement. X-rays show marked beaking of the vertebral bodies with narrowing of the intervertebral space. This condition may be associated with a posterior protrusion of the nucleus pulposus, in which case that complication would predominate and require whatever treatment was necessary on its own merits. There may also be associated changes in the apophysal joints. These would then probably account for the predominant symptoms.



Fig 447—Hypertrophic spondyloarthritis

Hypertrophic Spondyloarthritis

This is an involvement of the synovial joints of the vertebral column, which are mainly the apophysal joints, but there are also the tiny intervertebral synovial joints in the cervical region placed lateral to the

discs. Involvement of these joints is identical with that seen in other diarthrodial joints in the body. (See general section on Joints.) Histologically this is a true osteoarthritis. Degeneration of articular cartilage due to injury, rapid erosion due to overuse, slow erosion with the years, or vascular and metabolic disturbances predispose to or accelerate the process. The changes in the hyaline cartilage of the joint are quite marked before erosion to bone results. When there is bony contact, marked hypertrophic changes occur with the production of peripheral chondro-osteophytes. As one would expect from the pathological changes, symptoms are present long before there is x-ray evidence of hypertrophic change.

Clinical Features.—Pain and stiffness are often severe and characteristically influenced by the weather. In fact, in many patients their joints act as an excellent barometer. A fall in pressure is associated with an exacerbation of their symptoms. Symptoms are worse in the morning, but soon improve as the patients start moving about. They often discover for themselves that setting-up exercises before getting out of bed are beneficial. Symptoms are relieved by gentle exercise and aggravated by strenuous activities. The regions involved are proportional to the sites of maximal strain. These occur most frequently at the apex of the normal curves. Lumbosacral and lumbar regions account for two-thirds of the involvement, most of the others occur in the cervical region. Acute exacerbations of symptoms may produce marked muscle spasm. Reflex referral of pain occurs, according to the innervation of the part concerned. Novocain infiltration may be used to relieve muscle spasm or as a paraspinal block for both diagnostic and therapeutic purposes.

Treatment.—

1 General Measures.—

(1) Dietary directions may be necessary to deal with nutritional deficiency or alterations from the normal weight.

OSTEOARTHRITIS OF THE SPINE

Osteoarthritis of the vertebral column is generally taken to include traumatic and degenerative changes in the intervertebral and apophyseal joints. The two main types are *hypertrophic spondylitis* and *spondyloarthritis*.

Hypertrophic Spondylitis

This refers to changes in the intervertebral joints which are symphyses, and so the pathological changes differ from that seen in diarthrodial joints. Synonyms are *spinal osteophytosis* and *degenerative disc disease*.

teum at the attachment of these fibers and produces the hypertrophic reaction. The process is more marked anterolaterally than it is in the region of the anterior spinal ligament, presumably due to the protection that the ligament offers to the disc. The changes are absent or less marked posteriorly, because it is produced by a flexion mechanism. There is an associated reduction of spinal movement, and abnormal strains are thrown on the apophyseal joints; this may produce secondary degenerative changes there. Calcification of degenerated discs is occasionally seen.



Fig 446—Hypertrophic spondylitis.

The latter names one factor in a process which affects all the tissues of the joint. There is loss of the turgescence of the intervertebral disc, both with age and with degenerative processes. This dehydration of the disc is an important factor in its narrowing. As a result, there is interference with its function as a cushion between the vertebral bodies. Trauma to the adjacent bone then gives rise to peripheral ossification. This affects the bulging outer fibers of the annulus fibrosus. It probably lifts the periosteum

Clinical Features.—Hypertrophic spondylitis is occasionally present to a very marked degree and entirely symptomless. Examination will show, however, that there is a good deal of limitation of spinal movement. Very often there is a complaint of pain which is aching in character. Following strenuous exercise, or with the onset of fatigue, symptoms become more severe. If patients maintain one position for a prolonged period of time, it is difficult to start moving. After a short period of limbering-

up, there is a good deal of improvement. X-rays show marked beaking of the vertebral bodies with narrowing of the intervertebral space. This condition may be associated with a posterior protrusion of the nucleus pulposus, in which case that complication would predominate and require whatever treatment was necessary on its own merits. There may also be associated changes in the apophyseal joints. These would then probably account for the predominant symptoms



Fig. 447—Hypertrophic spondyloarthritis

Hypertrophic Spondyloarthritis

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Clinical Features.—Pain and stiffness are often severe and characteristically influenced by the weather. In fact, in many patients their joints act as an excellent barometer. A fall in pressure is associated with an exacerbation of their symptoms. Symptoms are worse in the morning, but soon improve as the patients start moving about. They often discover for themselves that setting-up exercises before getting out of bed are beneficial. Symptoms are relieved by gentle exercise and aggravated by strenuous activities. The regions involved are proportional to the sites of maximal strain. These occur most frequently at the apex of the normal curves. Lumbosacral and lumbar regions account for two-thirds of the involvement, most of the others occur in the cervical region. Acute exacerbations of symptoms may produce marked muscle spasm. Reflex referral of pain occurs, according to the innervation of the part concerned. Novocain infiltration may be used to relieve muscle spasm or as a paraspinal block for both diagnostic and therapeutic purposes.

Treatment.—

1. General Measures.—

(1) Dietary directions may be necessary to deal with nutritional deficiency or alterations from the normal weight.

(2) A regular exercise regime and regular hours of sleep should be established.

(3) Mental attitude should be improved by the avoidance of worry and fatigue. The cooperation of family and associates is necessary to create a cheerful atmosphere.

(4) Supplementary vitamin or hormone therapy is occasionally indicated

(5) Analgesics and sedatives are used as temporary expedients for rest and relief of pain

2. Local Measures —

(1) Postural defects are corrected by instruction in remedial exercises

(2) Heat and massage are used for their soothing and palliative effects during an acute exacerbation

(3) Body casts may be used for a short period to give support while building up musculature

(4) A Harris lumbosacral brace may be used to limit painful movement

(5) Manipulation is often dramatic in giving temporary relief. Instability or nerve root involvement must be positively excluded. The principle is to place the involved joints in the positions of the extremes of their movement, and then to stretch them slightly. The mechanism of relief is the snapping of light capsular adhesions

(6) Operative measures are worth while for severe and well-localized lesions. Occasionally an arthroectomy suffices and has the advantage of early mobilization. The vast majority, however, that come to operation require spinal fusion

(7) X-ray therapy is of doubtful value, but usually some temporary relief of pain is achieved.

Ankylosing Spondylitis

This condition is characterized by obliteration of the sacroiliac joints, ankylosis of the apophyseal joints, calcification of spinal ligaments and periphery of the intervertebral joints. It is often associated with involvement of peripheral joints. There are many

synonyms, *Marie-Strümpell spondylitis*, and *rheumatoid arthritis* of the spine being used fairly commonly. Others, such as *spondyloarthritis ankylopoietica*, and *spondylose rhizomelique*, are occasionally used. The disease follows a fairly characteristic pattern. Ninety per cent of cases occur between the ages of 15 and 35. It is sometimes seen in children, in association with Still's disease, which is childhood rheumatoid arthritis. It occurs in older persons as an exacerbation of the disease acquired during the usual age period. The cause of the disease is unknown. It is presumed to be an inflammatory process, as some focus of infection, recent or remote, can usually be established. There is probably an important allergic factor

Clinical Features.—The onset is slow, characterized by pain and stiffness. There may be reflex referred pain simulating other conditions. Once started, the disease appears to be progressive, but the rate is variable, anywhere from two to fifteen years, before it goes on to spontaneous remission. During this time there may be periods of acute exacerbation. The blood sedimentation rate is always high. Limitation of chest expansion and characteristic limitation of spinal movement are early features and may enable one to establish the diagnosis clinically before x-ray changes can be detected. The sacroiliac joint is involved in 90% of cases. It is spared only when the disease is restricted to the upper vertebral regions. It would appear clinically that the small joints of the spine are first involved, namely, the costovertebral and the apophyseal joints. This is followed by involvement of the sacroiliac joint which is the first one recognizable radiologically. This is followed by calcification in the spinal ligaments and the outer layers of the annulus fibrosus. When the process is advanced the x-ray acquires the "bamboo spine" appearance. About half the cases show peripheral joint involvement to some extent. It is unusual to have involvement of the small joints of the hands and feet. In fact, the hips and knees are

most commonly associated in the involvement. Histologically the process appears rheumatoid in nature. This does not explain the age and sex incidence, the response to treatment, the rarity of subcutaneous nodules, or the early calcification of peri-articular tissues. Acute cases, accompanied by fever, inanition and gross muscle wasting, may threaten life, but are fortunately rare.

consolidate the cure. Occasionally, in neglected cases, or if the deformity appears progressive, a Taylor brace may be of value. Commonly, with adequate treatment, it is unnecessary and if the disease is very active it is not well tolerated. A plaster bed with corrective felt pads may also be useful in the few cases that do not respond readily to x-ray therapy. Surgery is necessary in neg-

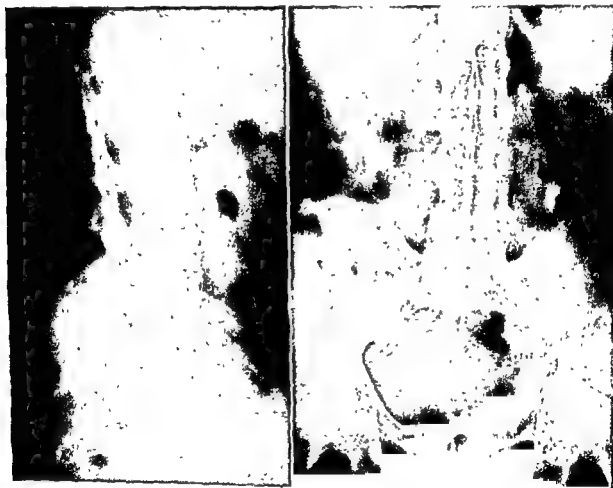


Fig 448—Ankylosing spondylitis

Treatment.—The general measures described for osteoarthritis of the spine are also applicable. X-ray therapy is of much greater value and in the majority of cases it completely arrests the disease. It produces an apparent improvement due to the abolition of pain and muscle spasm. Two or three courses of therapy are necessary, at about three monthly intervals, in order to

lect and late cases to correct deformity by osteotomy or by arthroplasty. The latter is most often indicated in the hip joint

Coccydynia

Coccydynia refers to pain in and around the coccyx and is therefore just a symptom-complex. It is often associated with trauma. There may be a fracture or there may be

traumatic arthritis in the sacrococcygeal joint. Congenital variations in the structure of the coccyx may resemble an old fracture. Pain may be due to a soft tissue inflammatory process either traumatic or infective in origin. The condition is commoner in females than in males. In some there are no localizing signs and the painful coccyx is presumably psychosomatic in origin. If there is displacement of the coccyx, it may be palpated externally, or by rectal examination, or demonstrated in x-ray. Sacrococcygeal arthritis shows well-localized tenderness, and aggravation of pain by movement of the joint. Pain with walking and exercise may indicate the trouble lies in the soft tissue attachments of the coccyx.

Treatment.—

- 1 Protection from pressure may be obtained by using an air cushion or sitting up straight on a hard chair. The weight is then taken on the ischium and the coccyx is relieved of any local pressure.

- 2 Short wave diathermy and massage are occasionally beneficial.

- 3 Novocain infiltrations into the site of maximum tenderness are worth a trial.

- 4 In selected cases with a clear-cut abnormality and failure of conservative treatment, excision of the coccyx may help. It is important to avoid operation in a neurotic patient.

Adolescent Kyphosis

This is a chronic disease producing a dorsal or dorsolumbar smooth kyphosis, commencing between the ages of 12 and 16 years. It may be symptomless or it may cause pain and fatigue. Occasionally, the pain and stiffness are quite severe. The synonyms are *Scheuermann's disease*, *vertebral epiphysitis* or *osteochondritis*. X-ray shows wedging of the vertebral bodies with quite a marked, smooth kyphosis. There appears to be fragmentation of the ring epiphysis and later on development of anterior marginal osteophytes. It has been thought that these

changes are due to a vertebral epiphysitis and osteochondritis. It is now, however, believed that it is entirely secondary to changes in the intervertebral disc. Schmorl demonstrated prolapse of the nucleus pulposus into the spongiosa of vertebral bodies in all his specimens of adolescent kyphotic spines, which came to dissection. This would result in loss of movement and tilting of the vertebrae. It is believed, therefore, that there are Schmorl's nodes at every level in the affected region. They are only seen in the x-ray if there is a reactive condensation of bone around the prolapse.



Fig. 449—Adolescent kyphosis

Treatment.—

1. Mild cases are treated by head traction, using a head halter with 20 pounds, for 20 minutes daily.

- 2 Intermediate cases may also require the use of a Taylor brace between exercise periods.

3. Severe cases may require recumbency for several months in a plaster bed with gradual correction by felt pads, along with hourly hyperextension exercises. All cases probably benefit from using a plaster bed at night.



Fig 450—Senile kyphosis

Senile Kyphosis

Senile kyphosis is probably due to degenerative changes in the intervertebral disc. There may be associated nuclear protrusion or annulus bulging, but these would not be etiological factors. There is a smooth upper and mid-dorsal kyphosis with narrowing of the intervertebral discs and a forward tilt of the vertebrae. The discs are thin and show loss of elasticity. There may be associated degenerative calcification in some of them. Some of the vertebrae are in contact anteriorly and show fusion of the spon-

giosa across the intervertebral disc. Senile degeneration alone would not account for the condition. There is probably an added postural defect or occupational stress. The texture of the bone is normal unless there is an associated defect.

Clinical Features.—Pain may be local or radiating in character and is often worse when lying down. It is self-limiting, disappearing as anterior fusion occurs. There may be a lot of distress in the intervening period. The remainder of the spine may be stiff. If compensatory curves cannot develop, the patient remains markedly stooped.

Treatment.—Heat, massage, and analgesics give symptomatic relief. Postural exercises are usually helpful. A Taylor brace in the day and a plaster bed at night are sometimes useful.

Senile Osteoporosis

Senile, idiopathic or postmenopausal osteoporosis, is characterized by backache and increasing deformity as vertebrae collapse. It affects middle or old age persons. The decalcification is not necessarily generalized. Often the vertebral collapse occurs in several regions. The x-ray shows lozenge-shaped intervertebral discs due to the turgescence of the disc causing a smooth bulge into the soft spongiosa. It is apparently a disturbance of bone metabolism.

Clinical Features.—There are loss of movement and moderate backache during quiescent periods. Vertebral collapse causes acute exacerbations. If there is root involvement, pain may be intractable. Every movement causes severe muscle spasm, and radiating nerve pain.

Treatment.

1. Chronic cases are treated by hyperextension exercises, plus a plaster bed at night, and a brace in the day.

2. Acute cases are treated by bed rest, with immobilization if necessary to relieve symptoms. Chordotomy for intractable pain may be considered.

3 High protein diet, vitamin "D," calcium Stilbestrol and Testosterone seem to help recalcification, but it is a slow process

Vertebral Osteochondritis

Vertebral osteochondritis (Calvé's disease) is an avascular necrosis in the vertebra. Presumably this is due to an infarct, but histological studies have not been made. It is in the same group of disorders as Kienbock's disease in the semilunar, Köhler's disease in the tarsal scaphoid, etc. It affects

adequate treatment it becomes reconstituted to appear normal. It is distinguished from Pott's disease by:

1. The intervertebral disc space remains normal.
2. There is no paraspinal abscess.
3. Absence of systemic reaction.
4. Negative tuberculin test.

Treatment.—Bed rest, in a hyperextension frame with four-point thoracic and pelvic traction, or a plaster bed with felt pads, is used till there is relief of pain and muscle



Fig. 451 —Senile osteoporosis

only one vertebra, usually lumbar or lower dorsal, in the 5 to 10 year age group. It is accompanied by kyphosis, pain, fatigue, tenderness, and muscular rigidity. The x-ray resembles Pott's disease of the rarer central body type. There is fragmentation with collapse of the vertebral body, but the appendages remain normal. There is tilting of the adjacent vertebrae, producing a sharp kyphosis, with the affected vertebral spinous process at the apex. The vertebral body may reduce to a wafer of bone, but with

spasm. A Castex corset, or spinal brace, is used until there is x-ray evidence of reconstruction of the affected vertebra.

Scoliosis

Structural scoliosis is a condition in which there is a lateral curvature of the spine, associated with altered shape and rotation of the vertebrae. It is important to separate the so-called *functional scoliosis*, which is really only a postural defect, and the *simple unprogressive structural* cases which are not

significant, from the *progressive structural* ones, which if untreated go on to a horrible deformity.

Early recognition and careful follow-up is the only way to select intelligently the few cases that require radical treatment. A glance at the flexed spine in all routine ex-

during a period of slow spinal growth, and then may progress rapidly and suddenly during a phase of rapid growth. Steindler refers to three periods of rapid growth:

1. Birth to 2 years.
2. 5 to 10 years.
3. Puberty to cessation.



Fig. 452.—Vertebral osteochondritis, before and after treatment

aminations at school age is imperative, and discloses even mild curves which should be noted and reviewed in three months. If there is any progression, a scoliosis record must be made. It is important to realize that there is, as yet, no substitute for continuous observation throughout the growth period. A curve may appear unprogressive

Closer observation is necessary at these times. When growth is complete, there is not likely to be any substantial change in the condition. In view of the fact that longitudinal growth of the vertebrae is by enchondral ossification, it is difficult to be sure just when it ceases. Risser has observed that ossification of the iliac apophysis

is an accurate index. It ossifies anteriorly to posteriorly, and as it curves around the posterior superior spine to point toward the sacroiliac joint, vertebral ossification is complete. This varies between 12 and 19 years, and indicates the preferable time for spinal fusion if that is necessary.

the iliac crests to occiput, each on one long film. A clinical photograph with the bony landmarks outlined is also taken. The curves are named according to the region and the side of their convexity. The apex and the limits of the curve are noted. The curve is measured by an empirical method. The



Fig. 453.—Congenital scoliosis showing measurement of primary and secondary curves

Method of Recording.—If the periodic observations are reduced to a simple formula, it leads to more accurate records, and is more easily understood if there are different observers. The x-rays should include anteroposterior views in the standing, sitting, and supine positions, extending from

last intervertebral surfaces still pointing into the concavity form the base line, and perpendiculars are drawn from these. The angle toward the axis of the spine is then measured.

Wedging is noted on a scale of 1 to 4 plus, measurements being made on the verte-

bra showing maximal wedging, and this is usually at the apex of the curve.

0 to $\frac{1}{16}$ = +

$\frac{1}{16}$ to $\frac{1}{8}$ = ++

$\frac{1}{8}$ to $\frac{1}{4}$ = +++

More than $\frac{1}{4}$ = ++++

Rotation of the vertebrae is also recorded where maximal, usually at the apex. It is positive if to the side of the concavity and negative if to the side of the convexity. It is measured by the relationship of the tip of the spinous process to the edge of the vertebral body.

To concavity To convexity

0 to $\frac{1}{3}$ = (+) (-)

$\frac{1}{3}$ to $\frac{2}{3}$ = (++) (--)

$\frac{2}{3}$ to edge = (+++) (---)

Beyond edge body = (++++) (----)

Usually there are three curves—the middle one being primary, and the other two are secondary. They are compensated when the sum of the curves to opposite sides is equal. The primary curve shows the most structural change and is the larger curve. Double primary curves are seen occasionally, being equal in all respects.

With high curves, *bending films* are necessary to determine the mobility of the curves. With lumbar scoliosis, this is done by the *pelvic tilt test*; the film being taken with a 3" lift under one buttock and then the other. If there is residual angulation in the compensatory curves, the total determines the limit of desirable correction in the primary curve. It is important to avoid overcorrection. Partial correction may be of remarkable value, if it restores compensation of the residual curves.

Types of Scoliosis

1. *Functional scoliosis* is a postural defect which can be voluntarily corrected. (See postural defects.) Adaptive changes in the structure of vertebrae are absent or minimal. This differs from scoliosis due to a short leg, or other deformities of the lower extremities. If the postural defect or the deformity of the

lower extremities is corrected, the scoliosis is no problem.

2. *Structural scoliosis* is due to an intrinsic defect in bone, nerve, or muscle. It cannot be voluntarily maintained in a corrected position.



Fig. 454.—Right dorsolumbar scoliosis

Classification according to etiology is customary and the approximate incidence is noted:

1. *Muscle* (a) Idiopathic—80%
(b) Thoracogenic—2%
2. *Bone* : (a) Congenital anomalies—3%
(b) Osteochondrodystrophy (Morquio's disease)—1%
3. *Nerve* (a) Poliomyelitis—10%
(b) Neurofibromatosis—2%
(c) Friedrich's ataxia—1%
(d) Spastics—1%

Clinical Features.—Very often scoliosis is unrecognized until someone remarks that there is a prominent hip, or a high shoulder or perhaps it is picked up in a school medical examination. Commonly it gives no symptoms in youth. So long as mobility and good muscular strength are present, compensation for the deformity appears adequate. Later there is apt to be degenerative change in the joints at the sites of maximal curvature. In very severe deformity, cord involvement with paraplegia can occur. With extreme curves there is secondary visceral embarrassment, which presumably shortens life. It is, therefore, imperative that the condition be treated before irreversible changes occur, with such dire consequences.

Treatment.—

1 Palliative.—

Exercises improve the sense of well-being and compensate for the ill effects of the curvature, but they do not appreciably or permanently alter the curve.



Fig. 455—The author's distraction brace

2. Corrective.—

(a) *Rest in recumbency* will produce appreciable correction. Albee used a Bradford frame with three canvas bands to give a cross pull against the curvature.

(b) *A distraction brace* gives traction on the spine between the pelvis and the occiput. It is limited by the pressure that can be tolerated on the chin and occiput; but is effective in very young children. The Milwaukee brace uses fixed distraction; the Butler brace uses four elastic distraction units attached by ball-and-socket joints to the headpiece, allowing some spinal movement.

(c) *Reverse curve plaster jacket* is used on the principle that pressure against the curve may alter growth by compression. It does not appear to do this to an appreciable extent.

(d) *Plaster casts* applied in corrective positions with and without crutch supports are of doubtful value. Often there is marked progression of the curvature in spite of such appliances.

3. Operative Treatment—It is generally recognized that a mature spinal fusion is the only means of maintaining a correction of structural scoliosis.

Opinions differ widely, but Cobb's indications for spinal fusion are:

1. A severe scoliosis usually over 50 degrees and correctable at least 20 degrees, preferably 12 to 21 years of age.

2. A lesser curvature which is increasing under conservative management and will become severe.

3. Unstable scoliosis, preferably at 13 to 14 years of age.

4. Pain which can be relieved by fusion; this is usually in the older patient.

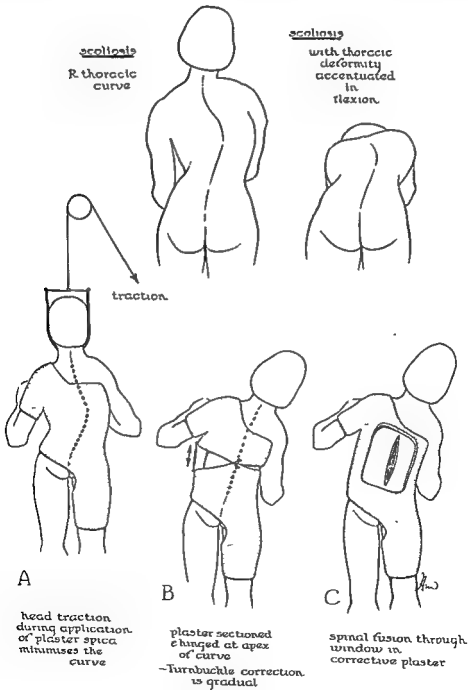
In some cases one is forced to fuse at an early age, in which case a short fusion is done as a compromise, and it is extended after vertebral growth ceases. This is more apt to be necessary in paralytic curves and in some congenital ones with multiple anomalies.

There is a variety of methods used for correction of the curvature prior to spinal fusion:

1. *Risser Jacket*.—A corrective cast is applied by

- (a) Head traction.
- (b) Albee Compere derotation table.
- (c) Other methods.

Hinges are incorporated back and front over the apex of the curve. Turnbuckles are attached and correction carried out as tolerated. When the desired position is obtained, the cast is reinforced and a large window is cut out over the spine for the fusion. This is preferred for high and rigid curves.



The Turnbuckle Corrective Plaster Jacket

2. *Transection Jacket*.—In dealing with double primary curves or mobile curves, the cast may be immediately sectioned and placed in maximal correction. The cast is reinforced in this position, the window cut, and operation proceeded with

3 *LeMesurier Hammock*.—This is an ingenious method using a fish net hammock. The patient is suspended by overhead pulleys in the hammock with suspension from the upper arm and leg as well, giving a four-point suspension. When the correction is considered adequate, a padded cast is applied. The posterior window can then be cut and operation proceeded with

The corrective jackets are usually retained for six months and some protection used for a further six months.

INFECTIOUS DISORDERS OF THE VERTEBRAL COLUMN

More than half of all bone and joint tuberculosis occurs in the vertebral column. By contrast, pyogenic infections of the spine are extremely rare, accounting for a very small proportion of all the cases of pyogenic osteomyelitis. Brucellosis, actinomycosis, typhoid spine, gonococcal, and syphilitic arthritis are all rarities

Tuberculosis of the Spine

Pott's disease may occur at any age but it is most common in the young. The involvement of the spine is secondary to tuberculosis elsewhere in the body, usually a tuberculous lymphadenopathy. Minor trauma, or a localized degenerative change due to the systemic toxemia, predisposes to an endarteritis, giving rise to the initial focus. In the spine, this commonly occurs in the vertebral body beneath the hyaline cartilage. This epiphyseal involvement accounts for two-thirds of the cases. The periosteum, central vertebral body, and appendages are involved with descending frequency. Subsequent events depend on virulence, resistance, localization, complications, and treatment. Caseation in the vertebral body per-

mits collapse, and expression of tuberculous debris forming a cold abscess. This may form

(1) A paraspinal abscess

(2) It may follow the course of muscles, nerves and blood vessels to reach long distances



Fig 457—Tuberculosis of L2 and 3 with collapse

(3) It rarely extends into the spinal canal giving rise to paralysis.

The cold abscess at a distance from the diseased area is found in about 20% of cases. It may account for a swelling anywhere between the occiput and the heel, but the commoner are:

1. *Retropharyngeal abscess*, arising in a cervical vertebra, perforating the preverte-

bral fascia, and pointing at the edge of the sternomastoid muscle.

2. *Intercostal abscess*, originating in a thoracic vertebra and following nerves or vessels.

3. *Psoas abscess*, from a lumbar vertebra, passing along the iliopsoas to the inguinal ligament on either side of the femoral artery.

sibly night sweats. Localization may be indicated by a tired ache, or tenderness, or the pattern of referred pain may indicate the nerve root involvement. Kyphosis, localized rigidity, tenderness or hyperesthesia may be noted. The x-ray in advanced cases shows collapse of the affected vertebral bodies and a paraspinal abscess. With early involve-



Fig. 458—Plaster bed for treatment of tuberculosis

Clinical Features.—The onset of the disease is often insidious, particularly in children who appear just out of sorts. The presence of a swelling, which investigation shows to be a cold abscess, or even the development of a sharp kyphosis, may be the first incidents which lead the individual to seek advice. Occasionally, low back pain, with acute sciatic radiation, occurs and closely simulates the symptoms of a prolapsed intervertebral disc. Inquiry elicits the symptoms of systemic involvement, loss of stamina, malaise, loss of weight and pos-

sibly night sweats. Localization may be difficult to interpret, but this is soon followed by decalcification, with a blurring of bony trabeculation. The hemogram may show increased sedimentation rate, lymphocytosis, and leukopenia. Muscle spasm is always present, and there is painful limitation of spinal movement. A young child will pick up an object by crouching on the hands and knees, instead of bending the back. Paraplegia is a serious complication which is not uncommon when the disease is in the dorsal region.

Treatment.—It is essential that treatment be correlated with an understanding of the disease in all its phases

(1) *Systemic treatment* by bed rest, controlled exposure to sun and wind, and intelligent diet are as fundamental as adequate immobilization of the diseased area. Mental health is achieved by education, occupational therapy, and an interesting environment,



Fig 459—Museum specimen showing tuberculosis of lumbar spine

with positive training in the philosophy of contentment. Supportive therapy, iron, vitamins, blood transfusions, etc., and antibiotics are used as required. The response to this regime is more effective in children than in adults, and therefore surgery as an aid to recovery is used accordingly

(2) *Arthrodesis* of the affected region achieves a more effective local immobiliza-

tion than external means, but does not in itself cure the disease. Delay for several months is essential in early disease to determine the number of vertebrae involved. Multiple involvement is a distinct possibility. Fusion is a delayed aid to recovery, only becoming effective when union is sound. One must be sure that the response to the general regime will be adequate for several months, to make the procedure worth while. Operation, when performed too early, increases the mortality rate; if too late, just prolongs the illness! Bradford frames or similar orthopedic appliances are commonly used to maintain the child in the position of hyperextension. Plaster beds with turning shells are adaptable to any age or condition. There are many methods of spinal fusion based on the original Albee and Hibbs procedures. The most popular are twin tibial grafts, and "H" shaped iliac grafts. When the systemic disease is arrested, and the vertebral lesion is recalcified, graduated convalescence is permitted. This consists of recumbency without plaster, increased exercises, sitting up in bed, and so on, to a gradual resumption of full activity over several months. Close supervision is required to guard against a possible relapse.

(3) *Streptomycin* control systemically and locally in wounds and sinuses has improved the scope of surgery in Pott's disease. Excision of the nidus of infection and other radical procedures are under clinical investigation and meeting with some success

There is a great divergence of opinion from that stated above as to the place of surgery in tuberculosis of the spine. Albee, with a very large experience, recommends fusion in any patient at any age, unless there is a poor operative risk, or there is fever due to secondary pyogenic infection. Some British surgeons, of great experience, only perform the operation when the disease is healed and if there is some mechanical defect. They would fuse to control instability—or to protect a well-healed lesion against trauma—

after a prolonged period of activity in a brace, without any sign of reactivation of the disease.

Sacroiliac Tuberculosis

Tuberculosis of the sacroiliac joints is not uncommon. It is chiefly seen in young adults and is serious if associated with multiple lesions elsewhere in the body. Multiple sinus formation is also an unfortunate characteristic in this region.

Clinical Features.—The diagnosis is usually missed until a painful limp develops. Pain may be localized to the sacroiliac region, but very often it is a diffuse low back pain. Occasionally it may be referred into the gluteal region and posterior aspect of the spine. A sciatic type of scoliosis is often seen. Either this or the muscle spasm in the gluteal region accounts for the limp. Abscesses are commonly seen and may present anywhere around the pelvis, most frequently pointing posteriorly over the joint. The usual systemic manifestations of the disease are present. X-rays show erosion of the joint surface, and a zone of decalcification around this, but in the early stages, it is difficult to detect any change.

Treatment.—Systemic treatment is the same as for tuberculosis elsewhere in the body. A sacroiliac joint is immobilized by the use of a plaster spica. If there are sinuses, a plaster bed is used. Streptomycin locally as well as systemically is very effective in obtaining healing of the sinuses and quiescence of the disease. This is followed by arthrodesis of the joint by one of the extra-articular methods.

Osteomyelitis of the Spine

Pyogenic infections of the vertebrae are most commonly due to *Staphylococcus aureus*. It is usually a blood-borne infection, rarely a complication of lumbar puncture, spinal operation, or injury. It may occur as a complication of septicemia in children, or it may localize in the vertebral body or the appendages of the vertebra, as the result of a

bacteremia. *Acute cases*, usually in the appendages, are characterized by fever, local tenderness, muscular spasm, and swelling of the overlying soft tissues. Oblique x-rays may show involvement of the interarticular joints. The *chronic type* is more often in the vertebral body, slow in onset, and resembling tuberculosis. Blood culture, hemogram, and tuberculin test often help to establish the diagnosis. The x-ray changes are late; they first show narrowing of the intervertebral space, and then collapse of the vertebral body. Subsequent sequestration and reactive changes in the bone, with thickening and bridging of the vertebrae, become characteristic. The complications of mediastinal abscess, empyema, suppurative pericarditis or extradural abscess may be serious.

Treatment.—Antibiotics are used in large dosage, combined with general supportive measures, such as blood transfusions and parenteral fluids. The spine is protected in a plaster cast or plaster bed. Abscesses may have to be drained, and if there is sequestration in an inaccessible location, drainage may persist. The hypertrophic reaction to the disease usually makes spinal fusion unnecessary, although it has been used in some of the chronic cases to give local support.

Gonorrheal Spine

Gonorrheal arthritis is not uncommon, but its occurrence in the spine is rare. It is suspected when there is pain and rigidity of the vertebral column following a Neisserian urethritis. The likelihood is increased if other joints are involved and if the complement fixation test is positive. It may produce a marked ankylosis.

Treatment.—The nidus of infection in the genitourinary tract must be eliminated first. Antibiotics, particularly penicillin, and the sulfonamides are of inestimable value. Local treatment of the spinal lesion may be symptomatic, with bed rest, followed by a plaster corset, until completely cleared.

Syphilitic Spine (See Fig. 321)

Syphilis is becoming a rare disease, and syphilitic involvement of the vertebral column is a great rarity. There is a variety of possible lesions.

- 1 Localized periostitis
- 2 Congenital syphilitic epiphysitis.
- 3 Syphilitic osteochondritis
- 4 Gummatous lesions
- 5 Extension from a syphilitic ulcer
- 6 Neurotrophic changes producing a Charcot spine

Cervical involvement is most likely by extension from a syphilitic pharynx. It is said to simulate Pott's disease. Other stigmata of syphilitic infection—Hutchinson teeth, nasal catarrh, suppurative otitis, interstitial keratitis, blood Wassermann, and x-ray appearance—help to establish the diagnosis.

Treatment.—The systemic disease is treated by penicillin and other antisyphilitic drugs as indicated. The local condition may require mechanical support. If there is instability, as with a Charcot spine, spinal fusion is indicated.

Other Infections of the Spine

1 *Typhoid Fever* sometimes produces a periostitis and osteitis of the spine.

2 *Undulant Fever* may produce abscesses in the vertebral body and intervertebral disc, or produce a hypertrophic periostitis. The general symptoms of the disease, plus Brucella agglutination, low white count, fever, and high sedimentation rate, aid diagnosis.

3 *Actinomyces* of the spine shows extensive bone destruction and numerous draining sinuses. Biopsy of the granulation tissue, with discovery of the ray fungus, establishes the diagnosis. Treatment is with iodides, surgical incision, and mechanical support to the spine.

4 *Blastomyces* resembles Pott's disease, the diagnosis is established by biopsy and microscopic findings. The prognosis is poor.

5 *Echinococcus* cysts of the spine require resection of the part, followed by mechanical support.

NEOPLASTIC DISORDERS OF THE VERTEBRAL COLUMN

Tumors of the vertebral column are rather rare. Those that do occur are mostly malignant and usually metastatic. Metastases occur from the breast, thyroid, uterus, gall bladder, and prostate, in descending order. The preponderant opinion is that the tumors are blood borne, the malignant emboli lodging in marrow capillaries.

Primary Benign Tumors

1. Osteomas
2. Osteochondromas—these are usually symptomless and no treatment is required.
3. Chordomas—the remnants of the embryonic notochord.



Fig. 460—Metastatic carcinoma of L2 with collapse.

4. Hemangiomas—usually in one vertebral body, common but rarely give symptoms. X-ray shows coarse trabeculation.

5. Dermoids, in connection with spina bifida occulta.

Primary Malignant Tumors

1. Fibrosarcoma—resembles Pott's disease but there is more rapid destruction

2. Endothelioma—this shows hematogenous metastases.

3. Multiple myelomas—these are recognized by plasma cells in a marrow biopsy, Bence Jones protein in the urine, and x-ray showing collapse of the vertebral bodies.

Metastatic Malignant Tumors

1. Carcinomas—there is gradual erosion of vertebrae with collapse.

2. Sarcomas—the erosion is more rapid.

3. Multiple myelomas.

The treatment of malignant tumors is symptomatic. Occasionally mechanical or operative support is indicated. Chordotomy is occasionally used to relieve severe pain.

REFERENCES

Albee, F. H., Powers, E. J., and McDowell, H. C.: *Surgery of the Spinal Column*, Philadelphia, 1946, F. A. Davis Company.

Blount, Walter, P., and Banks, Sam W., editors: *The American Academy of Orthopaedic Surgeons Instructional Course Lectures*, Vol. 5. Ann Arbor, 1948, Edwards Brothers, Inc.

a. Irwin, C. E.: *Paralytic Scoliosis*, p. 221.

b. Crowe, H. E.: *Introduction to Scoliosis*, p. 232.

■ Von Lackum, W. H.: *Surgical Treatment of Scoliosis*, p. 236

d. Risser, J. C.: *Important Practical Facts in Treatment of Scoliosis*, p. 248.

e. Cobb, J. R.: *Outline for the Study of Scoliosis*, p. 261.

Collins, Douglas H.: *Pathology of Articular and Spinal Diseases*, London, 1949, Edward Arnold & Co.

Fletcher, Ernest: *Medical Disorders of the Locomotor System*, Baltimore, 1947, The Williams & Wilkins Company.

Jameson, E. B.: *A Companion to Manuals of Practical Anatomy*, ed. 4, London, 1935, Oxford University Press

Merrill, Walter: *Orthopaedic Surgery*, ed. 4, London, 1950, Edward Arnold & Co

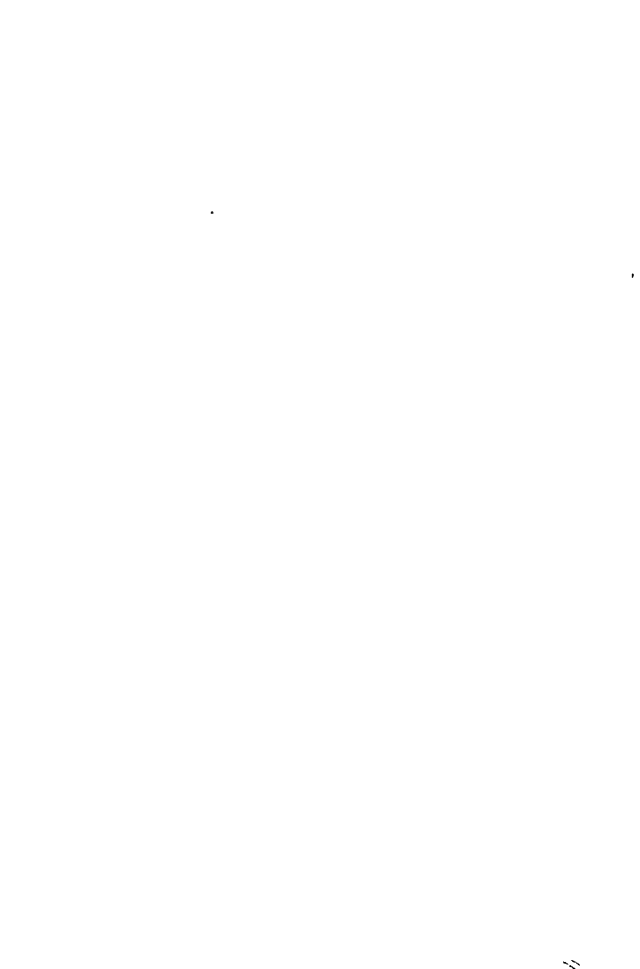
Shands, Alfred R., and Raney, R. B.: *Handbook of Orthopaedic Surgery*, ed. 3, St. Louis, 1948, The C. V. Mosby Company.

Speed, J. S., editor: *Campbell's Operative Orthopedics*, St. Louis, 1949, The C. V. Mosby Company

Steindler, Arthur: *Orthopaedic Operations*, Springfield, Ill., 1947, Charles C. Thomas.

Watson-Jones, Reginald: *Fractures and Joint Injuries*, ed. 4, Edinburgh, 1952, E. & J. Livingstone.

Wiles, Philip: *Essentials of Orthopaedics*, Philadelphia, 1949, The Blakiston Company.



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